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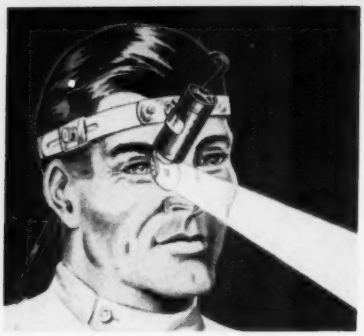

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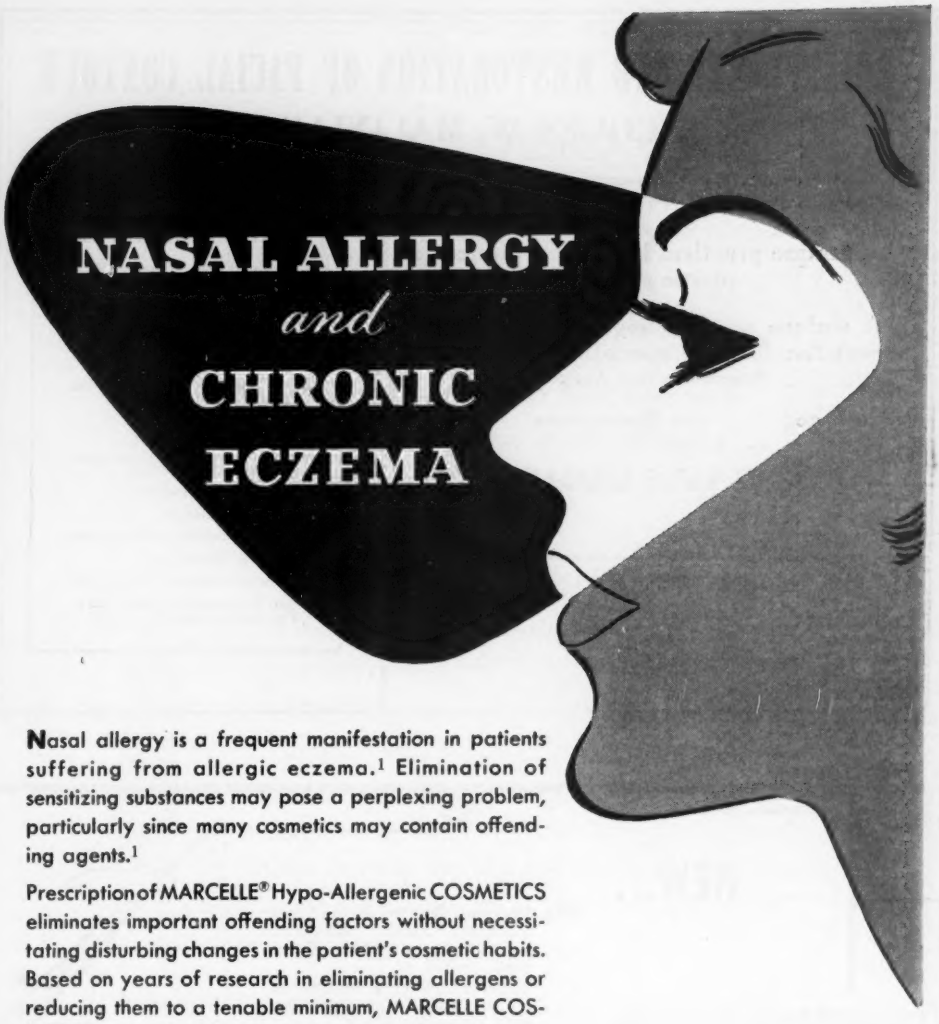

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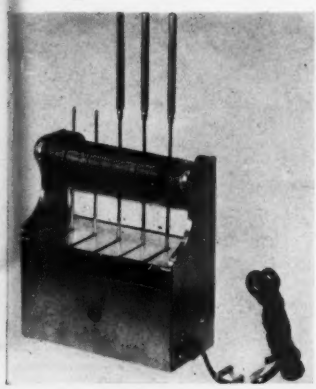

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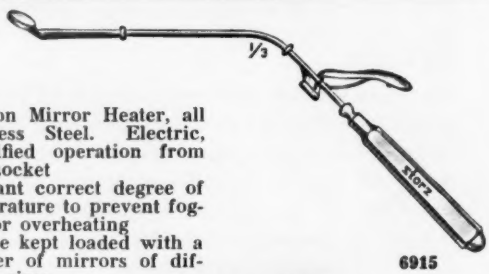

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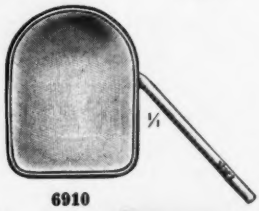

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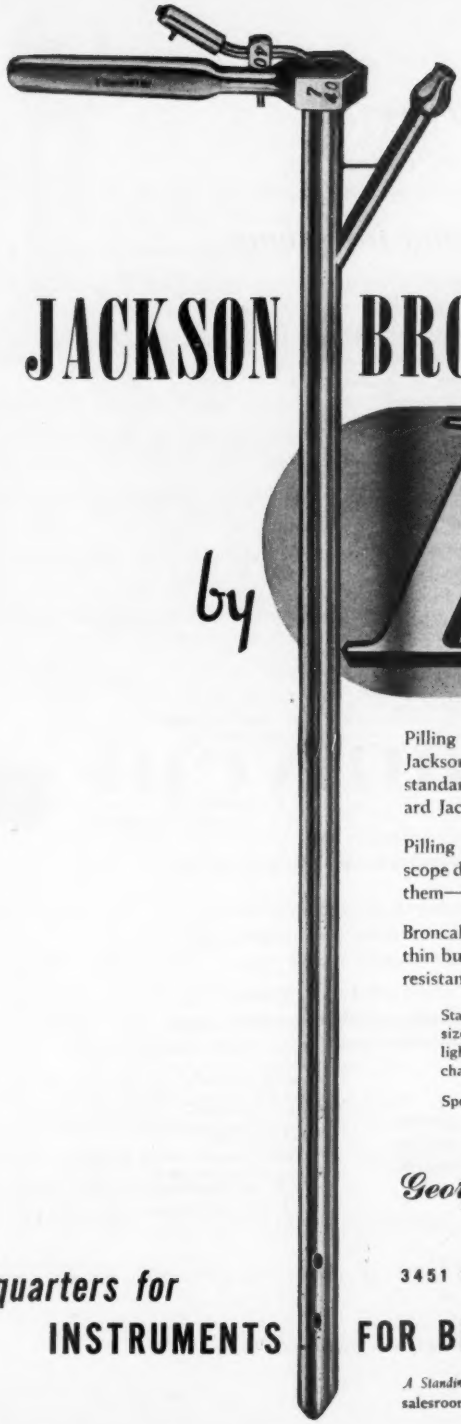

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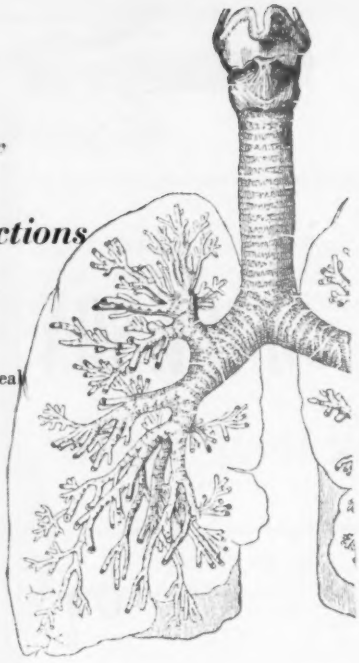

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CONTENTS

PAGE

Mouth of the Eustachian Tube: Action During Swallowing and Phonation. HENRY B. PERLMAN, M.D., CHICAGO 353

Observations on the Eustachian Tube. HENRY B. PERLMAN, M.D., CHICAGO 370

Treatment of Pulmonary Complications in Neurosurgical Patients by Tracheotomy. G. WINSTON TAYLOR, M.D., AND GEORGE M. AUSTIN, M.D., PHILADELPHIA. 386

Bronchoscopy in the Newborn: The Otolaryngologist's Contribution to Obstetrics. RAYMOND S. ROSEDALE, M.D., F.I.C.S., CANTON, OHIO 393

Juvenile Nasopharyngeal Angiofibroma. R. W. KERWIN, M.D., CHICAGO 397

Nonchromaffin Paraganglioma of the Middle Ear (Glomus Jugulare Tumor). J. S. LEWIS, M.D., AND R. N. GRANT, M.D., NEW YORK 406

Toxicity of Certain Topical Anesthetics Used in Otolaryngology: An Experimental Study. HENRY J. RUBIN, M.D., LOS ANGELES 411

Streptomycin in Otolaryngeal Tuberculosis. COLONEL A. J. VADALA, MEDICAL CORPS, ARMY OF THE UNITED STATES 421

Observations About the Presumed Action of Sodium Bicarbonate on the Otolithic Crystals. FABIO GIACCAI, M.D., AND ALDO CARLE, M.D., FLORENCE, ITALY 434

Incidence and Control of Trauma Accompanying Endotracheal Anesthesia: A Survey Based on the Response of One Hundred and Fifty Leading Laryngologists and My Experience of Twenty-Five Years in the Use of the Method. PALUEL J. FLAGG, M.D., NEW YORK 439

CASE REPORTS:

Scleroma of the Nose and Lip. LESTER W. EISENSTODT, M.D., NEWARK, N. J... 446

Cerebrospinal Rhinorrhea from Craniomastoid Fistula. RICHARD H. AMES, M.D.; WILLIAM D. FARMER, M.D., AND WARDELL H. MILLS, M.D., GREENSBORO, N. C. 451

Tumor of Carotid Body Type Presumably Arising from the Glomus Jugularis. JAMES L. POPPEN, M.D., AND P. A. RIEMENSCHNEIDER, M.D., BOSTON 453

Ossifying Fibroma of the Frontal Sinus. SIMON BALL, M.D., PHILADELPHIA... 460

CLINICAL NOTES; NEW INSTRUMENTS AND TECHNICS:

Plaster Skull Cap with Curved Arm Bumper Guard: Device for the Protection of Depressed Zygomatic (Malar) Bone Fractures Following Reduction. GERSON LOWENTHAL, M.D., CINCINNATI 466

PROGRESS IN OTOLARYNGOLOGY. Summaries of the Bibliographic Material Available in the Field of Otolaryngology.

Otitis Media and Complications. B. R. DYSART, M.D., PASADENA, CALIF... 468

NEWS AND COMMENT 481

DIRECTORY OF OTOLARYNGOLOGICAL SOCIETIES 482

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VOLUME 53 APRIL 1951 NUMBER 4

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MOUTH OF THE EUSTACHIAN TUBE

Action During Swallowing and Phonation

HENRY B. PERLMAN, M.D.
CHICAGO

INFORMATION regarding the physiological movements of the eustachian tube in man and the particular muscles involved in these movements has been obtained through anatomical dissection, observations on living animals and observations on living human subjects either by means of a nasopharyngoscope or directly through large pathological defects of the face and palate. These observations have not resulted in any general agreement about the action of the muscles in this region or the sequence and time relations of the movements that take place in man. Some representative studies and conflicting ideas are described below.

Cleland,[1] a contemporary of Toynbee, had a patient with an ulceration of the palate through which the mouth of the tube could be visualized. He taught the patient to swallow with his mouth open so that he could observe the tube at this time. He called attention to Toynbee's observations on the tube and Toynbee's conclusion that the tube is closed in the resting condition. Cleland's direct inspection of the tube led him to believe that the tube was open in the resting condition. He made some dissections of the levator and tensor muscles and stated that contraction of the superior constrictor during swallowing pressed the body of the levator against the lumen of the tube to close it at this time. He reported that pulling on the posterior fibers of the tensor palatine muscle produced a slight drawing inward of the wall of the tube so that "at the same moment when the faucial orifice of the tube is closed by the spasmodic elevation of its floor the commencement of the cartilaginous part of the tube has its outer and inner walls brought into momentary contact." He then went on to try to explain away Toynbee's observations in the following contradictory manner:

> Having now appealed to the anatomy of the parts as well as to observations on the living subject to disprove the theory that the Eustachian tube is usually closed and is opened in swallowing, we have yet to seek an explanation of the phenomena on which the theory is based, particularly the circumstance that when air is blown into the tympanum, displacement of the membrane continues until the act of swallowing is performed. The explanation that a film of moisture occludes the tube must have been forced upon [a subject with a cold. It is the correct one.] Propulsion of air through the nares acts powerfully in setting the moisture of the mucous membrane in motion and in the Eustachian tube it sends it back in the contrary direction to that in which it is normally carried by the cilia of the epithelial lining. The transverse diameter of the tube is very small and a very slight accumulation of moisture in any part of it will cause by capillary attrac-

From the Division of Otolaryngology of the University of Chicago.

This work was done in part under a grant from the Douglas Smith Foundation for Medical Research of the University of Chicago.

1. Cleland: On the Question Whether the Eustachian Tube Is Opened or Closed in Swallowing, J. Anat. & Physiol. **3**:97-103, 1868-1869.

tion an obstruction sufficiently strong to resist a considerable pressure of air or possibly may even cause its sides to adhere with the action of a sucker. The sudden closure and alteration in the form of the tube in swallowing and the equally sudden recovery of its natural form is precisely the operation most likely to burst such an obstruction, expel superabundant moisture and allow the escape of pent up air.

A few years later Yule [2] had a colleague watch his throat while he voluntarily opened his tube. No movement of the soft palate was noted, only a sustained medial movement of the posterior pillars that was controlled at will. He concluded from this and from his dissections that the tensor and levator did not act to open the tube. Only the active palatopharyngeus muscle opened the tube during swallowing. He commented also on the downward direction of the salpingopharyngeus in the resting tube and its role in keeping the tube normally closed. When the direction of this muscle was changed by the contracting upper curved fibers of the palatopharyngeus, the salpingopharyngeus could now on contraction move the posterior lip of the torus tubarius in such a way as to open the tube.

Zaufal [3] made observations on the function of the salpingopharyngeus muscle in a patient with a hole in the soft palate through which he could see this muscle fold. He was particularly concerned with the mechanism of closure of the nasopharynx rather than with the movements of the tube. He reported that the salpingopharyngeus moved less in phonation than in swallowing. He noted the elevation of the whole posterior wall of the nasopharynx during swallowing. He also noted that the torus was elevated to a more horizontal position during swallowing than during phonation. He stated that closure of the nasopharynx is weaker in phonation than in swallowing.

Joachim's [4] patient had a syphilitic defect enabling him to visualize the tube from above the palate. Joachim stated:

From the observations made on this patient I came to the conclusion that the tube opens during the act of swallowing, as it was possible to look deeply into the gaping ostium when the patient was examined for a length of time, during which the mucous membrane would become dry. In the position of rest the lips of the orifice of the tube are in contact in their whole extent, and the cleft is turned forward.

In 1920 Rich [5] briefly reviewed the conflicting opinions of earlier observers regarding the action of the tube and described his observations in the anesthetized living dog. The mouth of the eustachian tube was visualized through an incision of the palate, and the levator and tensor muscles, as well as the intemal pterygoid, superior constrictor and palatopharyngeus, were exposed by dissection. The tube mouth was found to be closed at rest and opened during swallowing. No other reflex mechanism, such as differences in pressure on the drum, mouth breathing, deep respiration or elevation of the palate, could cause the mouth of the tube to open.

By cutting experiments he found that the tensor was the only muscle that opened the tube. Cutting the tensor, however, had no visible effect on the movement of the

2. Yule, C. J. F.: On the Mechanism of Opening and Closing the Eustachian Tube, J. Anat. & Physiol. **8**:127-132, 1873-1874.

3. Zaufal, E.: Die Plica salpingopharyngeus, Arch. f. Ohrenh. **15**:96-144, 1879.

4. Joachim, O.: Contribution to the Physiology of the Soft Palate, Arch. Otolaryng. **18**: 226-234, 1889.

5. Rich, A. R.: Study of the Eustachian Tube: Its Related Muscles, Bull. Johns Hopkins Hosp. **31**:206-214 and 305-310, 1920.

soft palate. Cutting out the action of the levator, internal pterygoid, palatopharyngeus and superior constrictor had no effect on the reflex opening of the mouth of the tube during swallowing. However, cutting the levator did have an effect on movement of the soft palate. He repeated Politzer's experiments and found that the tensor was innervated by the fifth nerve and the levator by the tenth nerve and reported one human case in which unilateral failure to open the tube during swallowing in paralysis of the fifth nerve was seen with a nasopharyngoscope.

A related observation recently reported [6] indicates that there is a reduced tubal resistance to Valsalva's test in some patients after fifth nerve section for tic douloureux. Involvement of the motor fibers was suspected. There was no visible asymmetry of soft palate movement in these cases confirming observations of Rich that the levator is not supplied by the fifth nerve and that paralysis of the tensor has no visible effect on the soft palate. However, the tonus of the tensor may be responsible for tubal resistance to Valsalva's test. A minimal static pressure of 20 mm. Hg is needed to blow air through the tube into the middle ear by this method. Paralysis of this muscle would reduce the resistance of the tube to Valsalva's test and at the same time would explain the occurrence of fluid in the middle ear in some of these cases. The active physiological opening of the tube during swallowing may be impaired in these cases by the paralyzed tensor as Rich observed in his case and on dogs after cutting of this muscle.

In 1936 Worms, Coulouma and Varseveld [7] made careful dissections of the tubal area and were impressed by the intimate relation between the tube and the levator. They believed that the levator, lying under the entire length of the cartilaginous tube, moves all of it on contraction as well as dilating the tube mouth. They questioned the role of the tensor in opening the tube and called attention to the close relation of the tensor to the internal pterygoid.

In 1940 McMyn [8] made careful dissections on human cadavers of the muscles in relation to the tube, particularly with reference to the salpingopharyngeus muscle. He concluded that this muscle aids the superior constrictor muscles and the stylopharyngeus in raising the walls of the pharynx during swallowing. He did not think that the action of the palatopharyngeus had any effect on the patency of the tube but that, together with other muscles, it may slightly dilate the pharyngeal orifice of the tube. He thought like Yule that the contraction of the palatopharyngeal sphincter in swallowing might change the pull on the torus of the tube by the salpingopharyngeus from a downward to a posterior direction. He also concluded that the tube is opened mainly by the tensor and that the tube was closed passively. He found that the levator, unlike the tensor, was not attached to the tube. De Paula Assis in a more recent study [8a] found the salpingopharyngeus muscle in only 70 per cent of 50 dissections. He minimized the role of this muscle in occlusion of the nasopharynx but suggested that it might participate in opening the tube.

6. Perlman, H. B.: The Eustachian Tube: Abnormal Patency and Normal Physiological State, Arch. Otolaryng. **30**:212-238 (Aug.) 1939.

7. Worms, G.; Coulouma, D. M., and Van Varseveld: Considération sur la trompe fibro-cartilagineuse et la région pharyngo-tubaire, Bull. Soc. Anthrop. **8**:29-57, 1936.

8. McMyn, J. K.: Anatomy of Salpingo-Pharyngeus Muscle, J. Laryng. & Otol. **55**:1-22, 1940.

8a. de Paula Assis, J. E.: Observações anatômicas sôbre O musculus salpingo-pharyngeus, Rev. brasil. oto-rino-laring. **15**:169-186, 1947.

In 1943 Simkin [9] brought up the question of muscular activity about the tube, reporting on his dissections and observations in the living with a nasopharyngoscope. He placed considerable importance on the tension of the noncontracting salpingopalatine and salpingopharyngeus muscles as affecting closure of the tube at rest. He states that as the palate was elevated these muscles were released from tension. With depression of the pharynx the salpingopharyngeus actively pulls the attached tubal cartilage downward and laterally. He stated that with elevation of the palate the medial paratubal tissue is pushed upward and under the cartilage so as to unwind the cartilage from the slightly S-shaped course it follows toward the bony tube. He concluded that the tensor pushes the lateral tubal tissue into the lateral wall against the lumen, tending to keep it collapsed. With the release of tension in the salpingopalatinus and the salpingopharyngeus muscle the tube could open passively by inequalities of air pressure at the two ends of the tube. With the nasopharyngoscope he did not find a mucous blanket forming a plug at the ostium of the tube.

The monograph on the tube by Zöllner [10] in 1942 takes up the action of the tubal muscles in some detail. He calls the action of the levator one that moves the lower end of the cartilaginous tube up and back and names the muscle "musculus retrahens tubae." He observed that a succession of movements take place as the tube is carried back and up in line with the isthmus, while the tensor operates to open the tube for passage of air.

Gyergyay's [11] earlier direct observation using a spatula inserted through the mouth also points to a succession of movements rather than simultaneous movements of the tubal muscles in swallowing. Zöllner stated that there are no muscles that actively close the tube. The tube stays closed at rest because of its elastic properties and because of the pressure of the surrounding tissue. Zöllner describes the motion of the mouth of the tube during phonation and swallowing as seen with a nasopharyngoscope. Using the nasopharyngeal spatula of Gyergyay, he saw the sidewise motion of the tube due to the tensor but did not state that it was limited to either swallowing or phonation.

In 1943 Locchi and Castro [12] studied the mechanism of closure of the nasopharynx by dissection and fluoroscopic examination of the living. They describe a palatine fasciculus of the superior constrictor which would contribute to the medial displacement of the salpingopharyngeal fold in swallowing when that fold is driven as a wedgelike formation between the dorsal wall of the pharynx and the palatine velum.

Direct observations of the mouth of the tube in human subjects with large defects of the face with intact hard and soft palate are limited. The degree of exposure of

9. Simkins, C. S.: Functional Anatomy of the Eustachian Tube, Arch. Otolaryng. **38**:476-484 (Nov.) 1943.

10. Zöllner, F.: Anatomie, Physiologie, Pathologie, und Klinik der Ohrtrompete und ihrer diagnostisch therapeutischen Beziehungen zu allen Nachbarschaftserkrankungen, Berlin, Springer-Verlag, 1942, p. 54-60.

11. von Gyergyay: Direkte Untersuchung des inneren der knospeligen Ohrtrompete bis zum Isthmus, Ztschr. f. Hals-, Nasen-u. Ohrenh. **3**:362, 1922.

12. Locchi, R., and de Castro, M. J.: Mecanismo de fechamento do faringe nasal na deglutição a luz da disseccão e da cineradiografia, Rev. sudam. morfol. **1**:44-62, 1943.

the tube is usually restricted to a side view rather than a view directly into the lumen of the tube. Furthermore, concomitant disease may involve the tube itself to preclude normal movement.

Simpson and Witcher [13] recently studied five such cases by means of moving picture records. They found that even in phonation the soft palate and uvula are drawn upward, backward and toward the midline. There was a slight displacement of the torus upward and backward and of the region of the mouth of the tube especially downward and backward, but they saw no tendency toward enlargement of the lumen during phonation.

A yawn produced a palate movement similar to that of phonation but prolonged. However, in swallowing they found no change in the lumen of the eustachian tube in four cases and a slight widening of both the orifice and the lumen in the fifth. They concluded that the tube is open in the resting state.

They report that in swallowing, the soft palate and uvula have two movements: first, a very minor one consisting of slight elevation of the soft palate in the region of the uvula with the lateral walls of the pharynx below the tube being drawn toward the midline. This is followed immediately by the major movement in which the posterior third of the soft palate and the uvula are drawn about ½ inch (1 cm.) higher than the resting plane. The remainder of the soft palate is drawn downward, the lowest position being about ½ inch posterior to the attachment of the soft to the hard palate. The depression is across the whole extent of the soft palate but is most marked in the middle. After the initial depression, the palate is drawn upward, backward and toward the midline along with the lateral wall of the pharynx. The highest point of the soft palate in phonation or swallowing was 3 to 5 mm. below the level of the hard palate.

The role of the tubal muscles is also discussed in the recent monograph by Terracol, Corone and Guerrier.[14] While the action of the levator and tensor muscles is most commonly considered in a discussion of the movements of the palate and tube, the entire major process of nasopharyngeal occlusion that may affect the tubal area is accomplished by additional activity of the superior constrictor and the palatopharyngeus muscle. A recent report by Strong [15] on this problem supplements his earlier observations on closure of the nasopharynx and considers the muscles that can cause the lateral soft parts of the pharynx to move medially in assisting the closure. He calls attention to a group of lateral fibers of the palatopharyngeus arising from the upper surface of the palatine aponeurosis and passing out laterally through the inserting fibers of the levator to go into the inner surface of the superior constriction. He points out that a medial movement of the tubal cartilage and salpingopharyngeal fold is associated with this muscular activity and helps to obtain the precision in opposition of the occluding surfaces.

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13. Simpson, W. L., and Witcher, J. E.: A Study of the Eustachian Tubes, Their Orifice and Lumens in Patients with Large Operative Defects Giving Direct Visualization, Ann. Otol., Rhin. & Laryng. **56**:357-367, 1947.

14. Terracol, J.; Corone, A., and Guerrier, Y.: La trompe d'Eustache, Paris, Masson & Cie, 1949, p. 71-77.

15. Strong, L. H.: Function of the Salpingopharyngeus Muscle in Nasopharyngeal Closure, Anat. Rec. (supp.) **79**:58, 1941; Muscle Force Components in the Occlusive Mechanism of the Nasopharynx, ibid. **103**:510, 1949.

DIRECT OBSERVATION ON THE TUBE

In suitable subjects with defects of the face, motion pictures of the tubal area made with a magnifying lens at from 16 to 64 frames per second recorded frame by frame the motion sequences associated with phonation and swallowing. It is difficult to describe the details of these movements of the soft palate and tube observed in these subjects with unaided vision. When this motion is recorded in film and then projected frame by frame it is possible to reconstruct more clearly the sequence of events and the role that each part plays in the movement. Continuous movements of the palate and tube are observed in phonation. Phonation for every letter of the alphabet produces palate and tube movement. This movement at first glance resembles that seen during swallowing, but analysis of the frame by frame sequences shows important differences. In swallowing the duration of palate elevation is less than in phonation. For the letter "e" the palate is elevated for about 0.6 second. In swallowing the elevation lasts about 0.3 second. In phonation the soft palate rises and elevates the floor of the tube with it to a higher level than in swallowing, but the depth or lateral extension of the floor elevated is greater in swallowing (fig. 1). There is no associated lateral movement of the floor of the tube in phonation as there is in swallowing. In phonation, as the floor of the tube rises, only the posterior lip of the torus moves backward, upward and medialward. This movement obliterates Rosenmüller's fossa and the salpingopharyngeal and salpingopalatine folds. The apex of the torus tubarius does not shift and is the axis for the rising posterior lip in phonation. The mouth of the tube enlarges so that ½ inch below the apex the transverse diameter increases from an initial value of ¼ inch (0.5 cm.) to ⅝ inch (1.6 cm.) at the moment of greatest separation of the posterior lip of the torus. The posterior lip is elevated passively in a posterior and medial direction with elevation of the floor of the tube by the contracting levator of the palate. At the same time some active movement of the lower part of the posterior lip in the same direction is probably produced by the contracting and now medially elevated salpingopharyngeus muscle. In contrast to the posterior part of the tube, the anterior wall and anterior lip of the torus remain quite inactive in all this movement associated with phonation. The lumen of the tube at the mouth and deeper is usually bridged by a film of mucus that shows clearly in the moving picture record, and throughout the phonation activity, even with widening of the mouth of the tube, this mucus film remains intact (fig. 2). In one sequence Rosenmüller's fossa is closed completely for about 0.12 second by this posterior movement of the torus in phonation and reopens completely in about 0.1 second. The palate begins to drop first at its posterior edge; then this drop progresses toward the hard palate. Not only the palate but the floor of the tube rises higher on phonation than on swallowing. In the initial action of the levator during phonation the floor of the tube rises before the central part of the soft palate.

In swallowing, the central part of the palate and floor of the tube are not elevated so high as in phonation. In addition, there is a lateral (outward) movement of the floor of the tube. The period of widening of the mouth of the tube lasts about half as long as in phonation. The widening of the tube is produced not only by the posterior movement of the posterior lip of the torus into Rosenmüller's fossa but also by an anterior and lateral movement of the entire visible anterior wall of the tube (fig. 3). The movement of the posterior lip of the torus precedes that of the

anterior lip in the opposite direction by about 0.16 second. The anterior wall movement anteriorly reaches a maximum in about 0.12 second; then the movement is lateral for about another 0.12 second. There is a mass anterior and lateral movement of the anterior wall and anterior torus lip along with a similar movement of the posterior lip after the maximum opening of the tube mouth is observed. In another sequence, 0.2 second after the posterior lip of the torus has moved medially and posteriorly, the lower end of the anterior lip of the torus begins to move in an anterior and lateral direction to reach a maximum in about 0.12 second. Then the entire tube, both anterior and posterior lips, begins to move anteriorly and laterally for about 0.20 second, with the posterior wall moving more than the anterior wall,

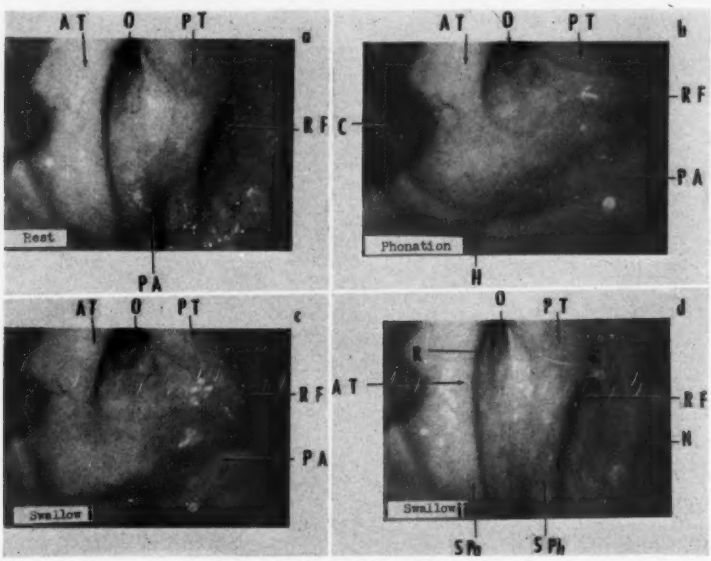

Fig. 1 (subject K).—View into the mouth of the tube and adjacent area. The top of the torus tubarius is left out in this exposure.

(*a*) Resting position with relaxed soft palate (*PA*), the opening into Rosenmüller's fossa (*RF*) being visible. In the resting position, the anterior (*AT*) and posterior (*PT*) lips of the torus tubarius extend below into the salpingopalatine (*S Pa*) and salpingopharyngeal (*S Ph*) folds (labeled in *d*). The mouth of the tube is seen at *O*.

(*b*) Same exposure during phonation, showing the elevated palate (*PA*) raising the floor of the tube and moving the posterior lip (*PT*) of the torus tubarius upward, backward and medialward, probably with the active participation of the salpingopharyngeus to occlude Rosenmüller's fossa (*RF*). The anterior lip (*AT*) of the torus does not move. The widening of the tube mouth (*O*) is seen. The junction between the hard and soft palate is seen at *H*. The posterior lip of the soft palate rises above that portion nearer the hard palate. *C* is the posterior tip of the inferior turbinate.

(*c*) Same exposure during swallowing, showing the mouth of the tube (*O*) widely open as the anterior lip (*AT*) and adjacent tube wall move anteriorly while the posterior lip (*PT*) of the torus tubarius moves in the opposite direction to close Rosenmüller's fossa (*RF*). The posterior lip of the palate (*PA*) rises above that nearer the hard palate. The palate, forming the floor at the tube mouth, is stretched and displaced laterally.

(*d*) Near the end of the action the palate has dropped away from the posterior wall of the nasopharynx (*N*) and the posterior lip (*PT*) of the torus tubarius has dropped to open Rosenmüller's fossa (*RF*). The anterior lip (*AT*) has returned to its resting position, but a ridge has formed (*R*) in the anterior wall just beyond the mouth of the tube (*O*) to narrow the lumen momentarily. The salpingopalatine and salpingopharyngeal folds are again clearly seen.

to reduce the lumen of the tube slightly and to change it into a more oval shape. As the palate rises, the posterior torus closes Rosenmüller's fossa in 0.1 second and a ridge on the elevated floor of the tube mouth moves posteriorly. The palate drops in 0.16 second to its original position with reopening of Rosenmüller's fossa; 0.25 second after the fossa is open again, the fold that has formed in the anterior wall of the tube just beyond the mouth has become most clearly defined. This fold then recedes completely in another 0.2 second. After the initial mass anterior movement of the tube, which leaves Rosenmüller's fossa wide open, the fossa is narrowed again, sometimes by a slight posterior shift. After 0.12 second of complete closure, Rosenmüller's fossa opens to the maximum in about 0.1 second, but after 0.2 second it

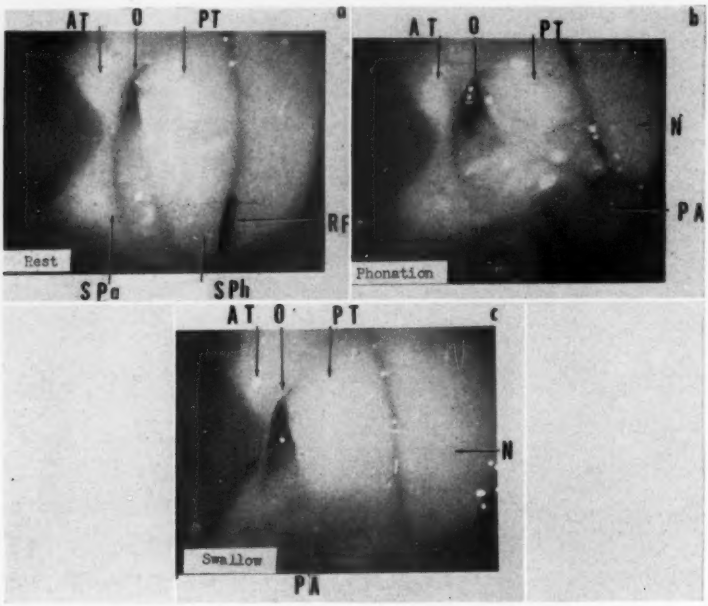

Fig. 2 (subject K).—Fairly direct view of the tubal area.

(*a*) Rest position with the palate down and the salpingopalatine (*SPa*) and salpingopharyngeal (*SPh*) folds in view. An opening into the lower part of Rosenmüller's fossa (*RF*) is visible. The mouth of the tube (*O*) is outlined by the anterior (*AT*) and posterior (*PT*) lips of the torus tubarius.

(*b*) The same exposure during phonation, showing the widening of the mouth of the tube (*O*) due to the movement of the posterior lip of the torus tubarius (*PT*) along with the elevation of the palate (*PA*). A film of mucus (see high lights) bridges the mouth of the tube. The posterior wall of the nasopharynx is seen at *N*.

(*c*) The walls of the tube are more deeply separated (*O*) in swallowing, and the floor of the tube and soft palate (*PA*) here are depressed and laterally retracted. The anterior (*AT*) and posterior lips (*PT*) of the torus tubarius move in opposite directions, bringing the anterior wall of the tube closer to the posterior edge of the inferior turbinate (pyramidal dark area on the left). Despite the deep separation of the walls of the tube, high lights show that the film of mucus across the lumen has not broken.

narrows a little because of this mass posterior shift of the tube. The film of mucus bridging the mouth, or lumen, of the tube is usually not broken even in swallowing (figs. 2 and 4), but sometimes this film is seen to disappear as the tube opens widely.

In another swallowing sequence, Rosenmüller's fossa is closed by the motion of the posterior lip of the torus in 0.11 second and remains fully closed for about 0.12 second. Then as the soft palate drops and the posterior lip of the torus tubarius returns toward its original position, the fossa begins to open during the following 0.08 second to reach a maximum in 0.16 second. Then the episode of the anterior canal wall ridge follows, to last about 0.4 second. This ridge actually narrows the lumen of the tube below the normal resting value. Just inside and ½ inch below the apex of the mouth of the tubal torus the initial separation of the walls of the tube of ¼ inch is increased to ½ inch at the moment of widest opening but is again

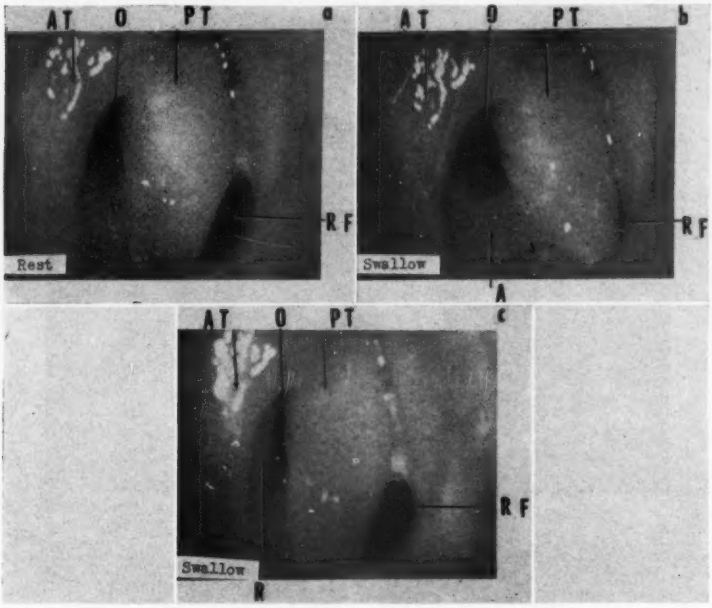

Fig. 3 (subject K).— Direct view of tubal area during swallowing.

(*a*) Rest position, showing mouth of tube (*O*) outlined by the edges of the anterior (*AT*) and posterior (*PT*) lips of the torus tubarius. An opening into the lower part of Rosenmüller's fossa (*RF*) can be seen because of the degree of the posterior lateral direction of the camera possible in this subject.

(*b*) Widening of the mouth of the tube (*O*) along with separation of the walls of the tube as the anterior (*AT*) and posterior (*PT*) lips of the torus tubarius actively move apart. The soft palate (*A*) in relation to the floor of the tube is stretched and retracted laterally. The movement of the posterior lip of the torus (*PT*) is greatest at its lower end, probably because of the active contraction of the salpingopharyngeus muscle, and almost obliterates the opening into Rosenmüller's fossa (*RF*).

(*c*) When the palate drops and the lips of the torus (*AT* and *PT*) return from their separated positions, a ridge (*R*) develops in the anterior wall of the tube to reduce briefly the size of the opening below normal. The opening into Rosenmüller's fossa (*RF*) is clearly seen.

reduced to ⅛ inch (0.3 cm) by the development of the fold of contraction in the anterior wall (fig. 3*c*). The active participation of the walls of the tube in changing the lumen is not seen in phonation (figs. 2*b* and 2*a*).

In swallowing, the mass shift of the tube after the mouth of the tube is widely opened is not only anterior but slightly downward. As the anterior shift begins, the lower elevated end of the posterior lip and salpingopharyngeal fold now drops to regain its normal position. In swallowing there is an initial widening of the mouth of the tube that is produced chiefly by the movement of this lower edge of the posterior lip (figs. 4 and 5). This movement is probably due to the active contraction of the simultaneously rising salpingopharyngeus muscle. Such separate motions of this cartilage may be possible because there are several small separate masses of cartilage in this region attached by connective tissue to the main mass of the cartilaginous tube. In phonation this active movement takes place along with the

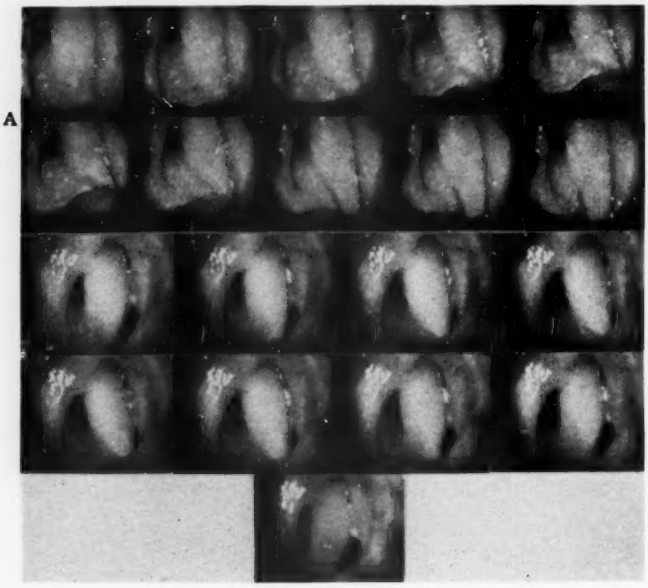

Fig. 4.—Two sequences of palate and tube motion in swallowing. *A*, the widened deep orifice of the tube (top row right) can be compared with that in the resting state (top row left). High lights of the mucous blanket show that it remains intact across the mouth of the tube even when widely open. After the initial widening, the tube lumen is narrowed (second row right) beyond that in the resting state by a ridge observed in the anterior wall of the tube. *B*, the separation of the lips of the torus tubarius with lateral displacement of the tube floor (third and fourth rows) opens the tube widely and deeply. A ridge in the anterior wall of the tube narrows the lumen at end of the sequence (bottom picture). The separate structures are identified in figures 1 and 2.

passive elevation of the entire mass of the posterior lip of the cartilaginous tube (fig. 6).

In 0.24 second after the onset of swallowing, the tube mouth may be maximally open, while the immediately following mass anterior shift and return to the original position may take another 0.24 second. As the central part of the soft palate is raised, probably by the action of the levator, the lateral portion at the mouth of the tube moves mostly in an outward direction, probably as the result of the action of the tensor muscle. The salpingopalatine and salpingopharyngeal folds are

obliterated. Some passive movement of the torus tubarius results. But early in this sequence one sees a special movement of the lower end of the posterior lip of the torus, probably as the result of the action of the rising salpingopharyngeus muscle. The following mass anterior shift of the tube as the soft palate drops is probably due to the unopposed contraction of the tensor veli palatini muscle. This tensor veli palatini activity in the second half of the tubal movement in swallowing may explain the ridge developed on the anterior wall of the tube that finally narrows the lumen (fig. 2 *c*). At what point in the sequence of movements is the tube lumen continuously open? Does it open during the time when the mouth of the tube is most widely open (at the beginning of the act), after the whole tube

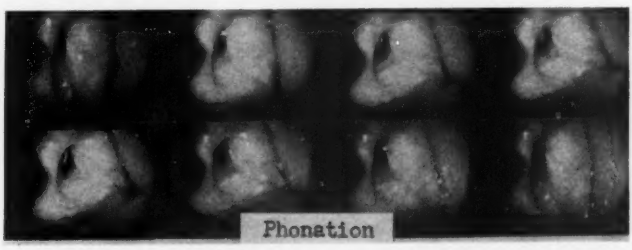

Fig. 5.—Sequence (starting at upper left) of tube and palate movement in phonation. Note the height of the posterior lip of the soft palate in the picture at the top right and the manner of the associated elevation of the floor of the tube. Widening of the tube mouth does not break the mucous blanket bridging this lumen (note the high lights reflecting from this surface). The various structures are further identified in figures 1 and 2.

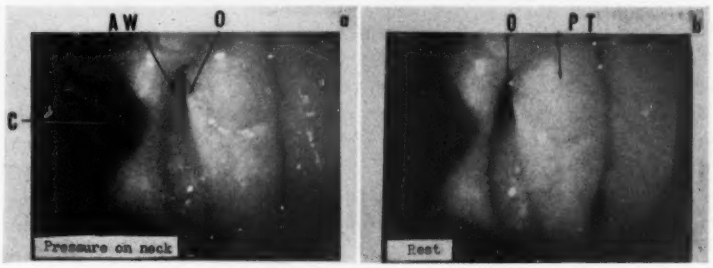

Fig. 6.—(*a*) Narrowing of the tube lumen by pressure on the neck. The lumen (*O*) is reduced to a slit (in comparison with that in *b*) as the soft anterior wall (*AW*) of the tube is pushed posteriorly against the hard and more slowly moving posterior wall. This procedure is often effective in relieving symptoms due to a continuously open tube. *C* is the posterior tip of the inferior turbinate.

moves anteriorly or during both of these events? The relation of tubal opening to the entire muscular activity of the swallowing act can best be determined by simultaneous recording of sound transmission through the tube and neck muscle action potentials and is reported in a separate paper. The duration of tubal opening during swallowing as measured in normal subject by sound transmission may be less than 0.2 second. The position of widest opening of the mouth of the tube as observed in the motion pictures is reached in about 0.25 second; then the duration of the anterior shift is about 0.25 second. The shape, duration and amplitude

of the acoustic envelope of the sound transmitted through the open tube is a better indication of the exact physiological function of the tube than direct observation of the lumen itself at the nasopharyngeal end. In swallowing, the posterior medial movement of the lower lip of the posterior torus seems to be the result of active contraction of the salpingopharyngeus muscle, since the floor of the tube is not elevated as in phonation (fig. 4). The lateral movement of the floor of the tube is probably due to the contraction of the tensor veli palatini muscle that also moves the anterior wall of the tube anteriorly. The lateral movement of the tube is not over when the posterior torus returns to the resting position and the contraction of the salpingopharyngeus ceases. The soft palate is still tensed and elevated, probably by the continued activity of the tensor. This is in contrast to simple elevation of the floor of the tube by the levator as seen in phonation (figs. 1 and 6). A fall in the palate after its initial rise in swallowing without breaking the seal between nasopharynx and pharynx may be in part the result of transient negative pressure developing in the pharynx when the cricoid sphincter opens. The drop in the level of the elevated palate may therefore be in part a passive one due to a relative increase in pressure from the nasopharyngeal side of the palate. The initial rise in the soft palate after hermetic closure of the pharynx has occurred may also be partly a passive phenomenon during swallowing resulting from the positive pressure built up in the oropharynx just before the cricoid sphincter opens but just after the soft palate has hermetically closed off the pharynx from the nasopharynx. A maximum pressure of 130 mm. Hg has been measured by Révész [16] in the oropharynx at this moment in the process of deglutition.

This pressure exerted against the under surface of the soft palate may cause some of the brief upward movement seen here in the motion pictures after the initial closure is accomplished by muscle action. The degree of this closure of the nasopharynx from the pharynx as accomplished by the musculature varies continuously in phonation as continuous changing palate movement is being produced by these muscles. This continuous variation on the shape and position of the soft palate during phonation indicates the great degree of cortical control of the pharyngeal musculature. However, the firmness of this closure in phonation is much less than in swallowing. While in phonation the soft palate can be blown away from the posterior pharyngeal wall by a positive intranasal pressure of about 10 mm. Hg, I found that during swallowing an intranasal pressure of about 100 mm. Hg is needed to break this seal.[17] The latter pressure is the same order of magnitude as the maximum pressure measured by Révész within the pharynx during swallowing. Analysis of another sequence of moving pictures during the act of swallowing shows that maximum opening of the mouth of the tube occurs as both lips of the torus tubarius move apart within about 0.2 second; 0.16 second later the lumen returns toward its original size by the return of the posterior lips while the anterior lip remains deviated. In another 0.16 second there is a maximum anterior shift of the mouth of the tube with return in 0.16 second to the original position. Then the fold in the anterior wall of the tube is most marked, receding in the following 0.16 second. This indicates activity lasting about 0.85 second.

16. Révész, G.: Die Rolle von intraoralen Druckfactoren in der Pathologie der Tonsillen, Pract. oto-rhino-laryng. **11**:193-205, 1949.

17. Perlman, H. B.: Quantitative Tubal Function, Arch. Otolaryng. **38**:453-465 (Nov.) 1943.

In swallowing, the inferior part of the posterior torus is moved and thickens the fold of the raised salpingopharyngeus muscle before there is a change in the salpingopalatine fold (figs. 4 and 5). The latter is obliterated when the palate is at maximum height and tension during swallowing. In the extreme elevated position, the posterior edge of the palate is higher than the anterior edge just medial to the tubal orifices (fig. 1). The ridge in the floor of the tube near the posterior torus remains high after the edge of the soft palate begins to drop away from the posterior wall of the nasopharynx and falls below the level of this ridge. Then the ridge drops with the floor as the salpingopalatine and the salpingopharyngeal fold reappear. The soft palate rises, and the posterior movement of the posterior lip

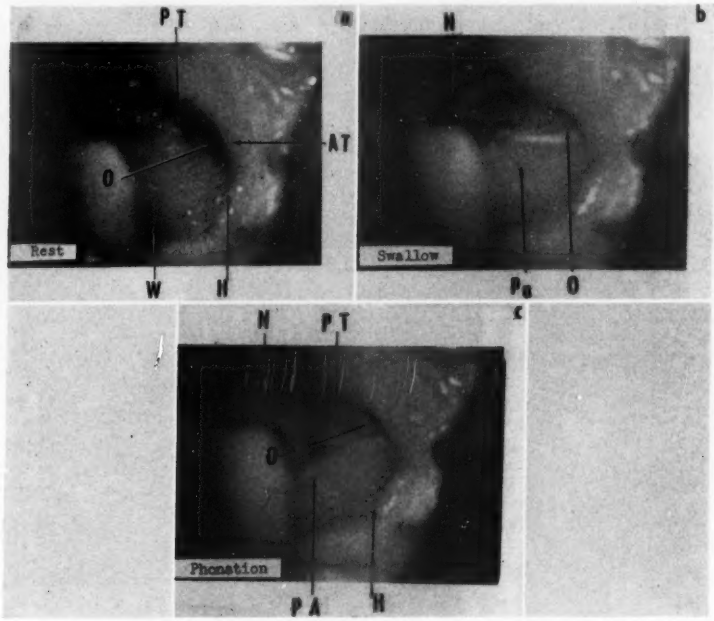

Fig. 7 (subject R).—Oblique view of the tubal area.

(*a*) Rest position. The orifice (*O*) of the tube is barely visible. The anterior lip (*AT*) of the torus tubarius is seen in relation to the posterior edge of the hard palate (*H*). The posterior wall (*W*) of the pharynx is visible because the soft palate is not elevated.

(*b*) Position during swallowing. The elevated soft palate (*Pa*) is opposed to the posterior wall of the nasopharynx (*N*). The movements of the lips of the torus tubarius are difficult to see because of the oblique view.

(*c*) Position during phonation. The mouth (*O*) of the tube is seen in relation to the elevated soft palate (*PA*) and the posterior, medial and superior displacement of the posterior lip (*PT*) of the torus tubarius. The posterior wall of the nasopharynx is seen at *N* and the junction between the hard and soft palate at *H*.

of the torus closes Rosenmüller's fossa before the ridge appears on the upper surface of the palate in and near the floor of the tube. Both salpingopalatine and salpingopharyngeal folds are simultaneously obliterated by the rising soft palate and tube floor during phonation. The salpingopharyngeal fold changes more than the salpingopalatine fold. In swallowing, the anterior movement of the anterior

lip may start about 0.04 second after the posterior lip begins to move in the opposite direction. As the posterior lip returns from its deviated position, the palate drops and the transverse tension on the floor of the tube recedes as the lips of the torus tubarius come together. The palate is down when the anterior shift of the tube takes place during swallowing and does not rise again. The ridge in the floor of the tube may be the result of a contracted bundle of levator fibers, since it is also seen in phonation (figs. 4 and 6), when it goes higher with the rising palate and is closer to the lower edge of the posterior lip. In one sequence Rosenmüller's fossa was closed for about 0.56 second during phonation but only 0.33 second during swallowing.

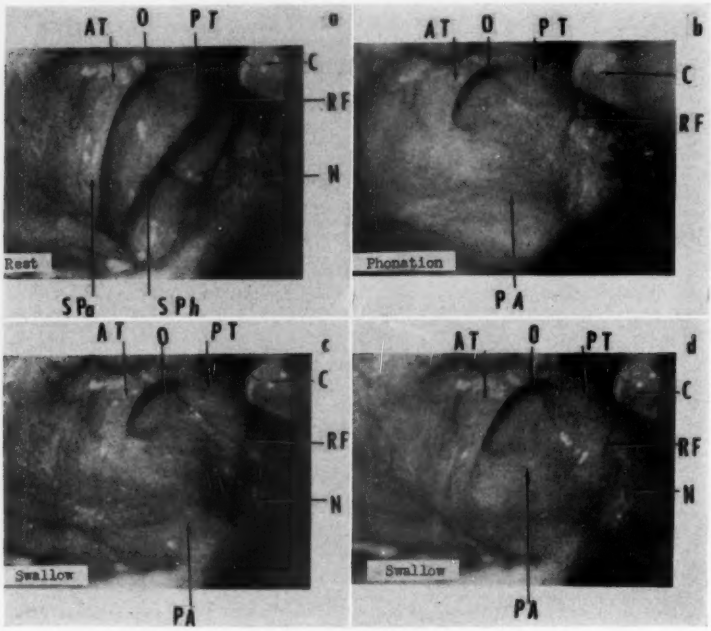

Fig. 8 (subject B).—Oblique view of tubal area.

(*a*) Tube and palate at rest. The mouth (*O*) of the tube is outlined by the anterior (*AT*) and posterior (*PT*) lips of the torus tubarius. The salpingopalatine (*SPa*) and salpingopharyngeal (*SPh*) folds are clearly defined. The opening into Rosenmüller's fossa (*RF*) is seen in relation to the posterior wall of the nasopharynx (*N*). C is the posterior tip of the inferior turbinate.

(*b*) The same view during phonation showing the elevated soft palate (*PA*) closing the nasopharynx and displacement upward, backward and medialward of the posterior lip (*PT*) of the torus tubarius to widen the mouth of the tube (*O*) and close the opening into Rosenmüller's fossa (*RF*). The anterior lip of the torus tubarius does not move.

(*c*) The same view during swallowing shows the elevated soft palate (*PA*) apposed to the posterior wall of the nasopharynx (*N*). The anterior lip (*AT*) and posterior lip (*PT*) of the torus tubarius move in opposite directions to open the tube mouth (*O*).

(*d*) A moment later one sees the further widening of the tube mouth (*O*) with lateral displacement and stretching of the soft palate (*PA*) in the floor of the tube.

In addition to the movement of the tube and palate, there is also activity in the areas probably occupied by the upper edge of the superior constrictor

muscle, the posterior pharyngeal wall and the posterior wall of Rosenmüller's fossa. The latter areas are seen to move in swallowing. The posterior wall of Rosenmüller's fossa moves anteriorly when the mass anterior shift of the tube takes place. Then as the tube moves back, this wall moves back and medially and thereafter regains its original position. With the initial posterior movement of the torus this wall moves in a similar direction. Little if any activity is seen in this area of the nasopharynx during phonation. In phonation, the medial movement of the posterior walls of the pharynx with the creation of Passavant's ridge is more suggestive than in swallowing. In swallowing, as the levator is going up it causes the posterior lip of the torus and tube wall to move posteriorly into Rosenmüller's fossa while the tensor is pulling the anterior wall anteriorly. Then as the levator contraction begins to subside and the palate drops, the tensor works without

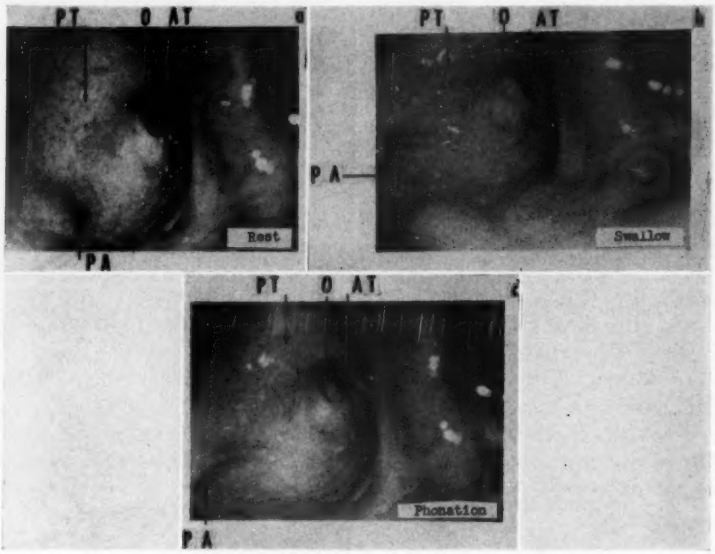

Fig. 9 (subject M).—Syphilitic scarring of the tubal area.

(*a*) In the rest position the soft palate (*PA*) hangs down and the mouth of the tube (*O*) is small due to the apposition of the scarred lips (*AT* and *PT*) of the torus.

(*b*) The anterior (*AT*) and posterior (*PT*) lips of the torus tubarius are scarred and their motion is limited. The change in the mouth of the tube (*O*) is small even in swallowing. The elevated soft palate (*PA*) is now apposed to the posterior wall of the nasopharynx. There is some stretching and lateral movement of the soft palate in relation to the floor of the tube.

(*c*) Same area during phonation. The mouth of the tube (*O*) is more oval than in (*a*), and the deformed torus has restricted movement. The soft palate (*PA*) is apposed to the posterior wall of the nasopharynx.

opposition to move the whole tube anteriorly after the palate drops. The lumen is widest just before the palate begins to drop. The posterior torus is elevated passively by the contracting levator, and the lower lip of this torus is probably moved back, up and medially by the salpingopharyngeus. The anterior torus and wall are pulled actively in the anterior direction by the tensor veli palatini muscle.

As Rich observed in animals, no reflex activity of the tubal muscles was elicited in these subjects by manipulations of the ear. Narrowing of the tube lumen could

be demonstrated when moderate pressure was applied on the neck just below the angle of the mandible. This pressure produces a mass shift of the entire visible tube into Rosenmüller's fossa as well as an additional relative movement of the soft membranous anterior wall against the firm posterior cartilaginous wall of the tube. The oval lumen was seen to change into a narrow slit near the posterior wall by this maneuver (fig. 7). This is visible evidence of the previously reported increased resistance of the tube by this maneuver and its ability to relieve symptoms of autophony and other conditions in patients with an abnormally patent tube.

Other examples of tubal activity during swallowing and phonation recorded by the camera in this study are seen in figures 8 to 11. These scenes are from subjects that are less favorable for analysis of normal tubal activity because the face

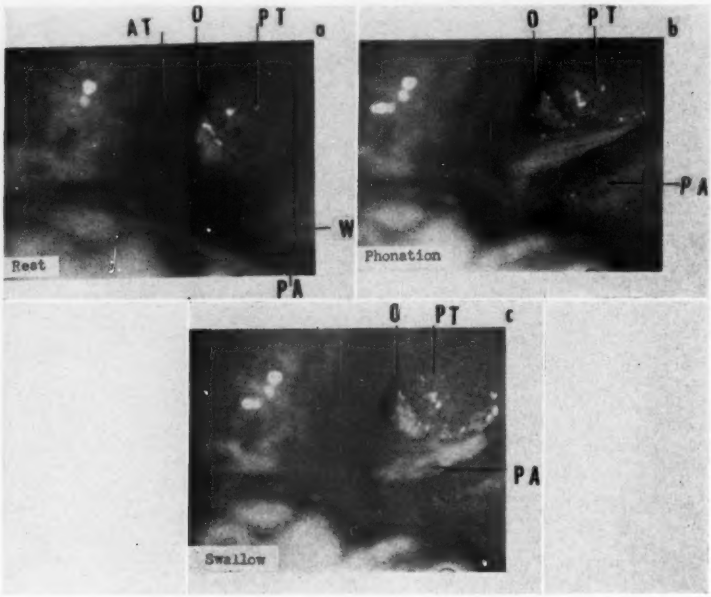

Fig. 10 (subject M).—Side view of area of the nasopharynx which is free from syphilitic scarring.

(*a*) Rest position showing mouth of the tube at *O* in relation to the anterior (*AT*) and posterior (*PT*) lips of the torus tubarius and the posterior wall of the pharynx (*W*) which is visible because the soft palate (*PA*) is down.

(*b*) Same area during phonation with elevation of the soft palate (*PA*) and floor of the tube together with a posterior, superior, and medial movement of the posterior lip of the torus tubarius and widening of the mouth of the tube (*O*).

(*c*) In swallowing the elevated soft palate (*PA*) is also retracted laterally (end of arrow) but the oblique view of the tube obscures the action within.

defect was not great enough to permit a view directly into the tube (figs. 8 and 9) or this region was abnormal. One subject's face had been destroyed by syphilis, and the syphilitic scarring involved the left tubal area. The appearance and movements of this tube (fig. 10) were in contrast to those of the opposite uninvolved side (fig. 11).

SUMMARY

A historical review of the observations on the physiological movements of the eustachian tube is presented.

Four subjects with defects of the face, in whom the tubal area could be observed directly from above, were studied by means of moving pictures. These pictures, taken at 16 to 64 frames per second through a magnifying lens, were projected frame by frame. In this way the sequence of movements of the different parts could be followed and timed.

A great deal of activity of the soft palate and the adjacent tubal area is seen in phonation and swallowing. In phonation, only the posterior lip of the torus is seen to move. This movement is backward, upward and medially as the soft palate and floor of the tube are elevated. The posterior lip appears to be lifted passively by the contracting levator and separates the edges of the tube mouth. Active movement by the simultaneously rising salpingopharyngeus also contributes to the observed additional displacement of the posterior lip in its lower portion.

In swallowing, the anterior and posterior lips of the torus actively move in opposite directions while the soft palate is elevated and retracted laterally to include that portion forming the tube floor. The anterior wall of the tube enters into this anterior movement along with the torus, and the resultant opening of the mouth of the tube is greater, deeper and rounder than on phonation. Then there is a mass anterior shift of the tube. After the initial return of the separated walls of the tube, a ridge forms in the anterior wall that momentarily narrows the lumen below that of the resting state.

The lumen of the tube may be bridged by a mucous blanket during phonation and even during swallowing. Even when this mucus is not seen bridging the visible lumen near the mouth of the tube one cannot determine by direct vision the condition of the tube lumen extending into the middle ear through the fixed or bony portion of the tube. Other methods than direct inspection of the mouth of the tube, such as the study of sound transmission through the tube, are necessary to determine whether the resting tube is normally closed or open and what changes in the continuity of the air molecules in the middle ear with those at the mouth of the tube occur during physiological action.

The tube lumen may be narrowed by pressure on the neck.

No reflex activity of the tubal muscles could be initiated by manipulations of the ear.

OBSERVATIONS ON THE EUSTACHIAN TUBE

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ON PERUSAL of current literature one notes that despite a great deal of clinical observation on the eustachian tube, the understanding of its physiological role as a ventilator of the middle ear is limited. To broaden the knowledge of this function in man under ordinary conditions, more experimental observations are needed. These have been difficult to devise, since the function of the tube as a ventilator of the middle ear is difficult to detect. Excepting the reports on air pressure changes and the tube by Hartmann [1] and by Armstrong and Heim,[2] basic experiments in man are few. The observations by Politzer [3] and by Lucae [4] with a monometer sealed in the external canal showed the type and degree of pressure changes transmitted to the middle ear when the tube opened while swallowing with the nose closed (Toynbee's maneuver). Politzer also called attention to the ability to hear sound transmitted from the nose to the ear when swallowing as proof of tubal opening. Observations on the nature of this sound transmission as an indication of tubal opening have been limited. Kiml [5] recently questioned the significance of this subjective increase in loudness as an indication of tubal opening.

Gyergyay,[6] using a pneumomassage apparatus in the nose, recorded, with a Maryes diaphragm in the canal, the vibrations transmitted to the ear through the open tube during swallowing. By auscultating the ear, he observed that a sound at the nose was only infrequently heard at the ear during swallowing by a normal person. This sound transmission changed in the same person from time to time and differed from one person to another. He considered that a tube that opened every time one swallowed was pathological. The tube was found at times to transmit some sounds better than others.

From the Division of Otolaryngology of The University of Chicago.

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1. Hartmann, A.: Experimentelle Studien über die Funktion der Eustachischen Röhre, Leipzig, Veit Comp. 1879.

2. Armstrong, H. G., and Heim, J. W.: The Effect of Flight on the Middle Ear, J. A. M. A. **109**:417 (Aug. 7) 1937.

3. Politzer, A.: Über subjective Gehörsempfindungen, Wien. med. Wchnschr. **15**:1710, 1865.

4. Lucae, A.: Zur Function der Tuba eustachii und des Gaumensegels, Arch. f. Ohrenh. **3**:179, 1866.

5. Kiml, J.: Politzer's Acoustic Method of Examination of the Permeability of the Eustachian Tube: A Criticism, Excerpta med. XI **3**:262, 1950.

6. Gyergyay, Á.: Neue Wege zur Erkennung der Physiologie und Pathologie der Ohrtrompete, Monatsschr. f. Ohrenh. **66**:769, 1932.

EXPERIMENTAL METHOD

To obtain objective quantitative data on some of these acoustic phenomena described above, an experimental setup was used that was similar to that used previously in the study of normal and abnormal patency of the tube.[7]

The subject was seated comfortably in an electrically shielded quiet room. An ear tip with a short plastic tube was placed in his external canal and coupled to a calibrated condenser microphone. To record the throat muscle activity during swallowing, an electrode was pasted to each side of the neck at the level of the hyoid bone. A hand signal was sometimes provided for the subject, so that he could record the time when he swallowed or opened his tube voluntarily. A 20 or 100 cycle timing wave was introduced to the recording apparatus for time measurement. The amplitude from the microphone was set so that the base line was quiet when no sound was coming from the subject. When required, a low frequency sound source (100 or 200 cycles) was delivered to the subject's nose through tubing from a loud speaker driver unit. This sound delivered to the nose was not recorded, nor was that of phonation at the mouth. In the initial experiments the relatively low speed of movement of the bromide paper in the General Electric loop oscillograph did not permit accurate recording of the high frequency components of the acoustic disturbances taking place in the subject's external canal. The acoustic interaction between the diaphragm of the microphone coupled through tubing to the ear canal, and the moving parts of the subject's ear (drum, etc.) precluded any finer analysis of the exact wave shape of the transient acoustic phenomena generated within the subject. Later experiments were carried out with the use of a calibrated condenser microphone and recording either from galvanometers on fast film or from the cathode ray oscillograph. These permitted a more accurate evaluation of the various acoustic phenomena, including the rapid transients. The over-all change in the acoustic envelop was monitored on the oscilloscope as a criterion for change in the amplitude, etc., that resulted from changing the nature of the acoustic pathways for sound in the nasopharynx as they reached the external canal and were recorded by the microphone coupled to it. The width (amplitude) of the acoustic envelop when humming with the tube ordinarily closed was made small in order that the galvanometer could record without distortion the greater amplitude resulting when the tube was open. No change in the amplification was made after the recording was once started.

Care is taken to maintain otherwise similar acoustic conditions in these experiments, i. e., the mouth is closed and one nostril remains open. The glass tip for the external sound source is fitted snugly into the opposite nostril. A 100 cycle sound source was found more satisfactory for an experiment than one of higher frequency, i. e., 250 cycles. With this external sound source it was definitely shown objectively and subjectively that the tube often does not open, especially when one is swallowing voluntarily without something in the mouth. To obtain definite opening in the experiment the subject usually put some water into his mouth and swallowed it on signal from the operator of the recording equipment. Similarly it was evident that despite a great deal of movement of the soft palate and mouth of the tube during phonation, the tube did not open during phonation to transmit an external sound source at the nose. The experimental setup for studying acoustic phenomena associated with a voluntary opening of the tube during speech and with sound delivered to the nose from an outside source is illustrated in figure 1 *A* and *B*.

7. Perlman, H. B.: The Eustachian Tube: Abnormal Patency and Normal Physiologic State, Arch. Otolaryng. **30**:212-238 (Aug.) 1939.

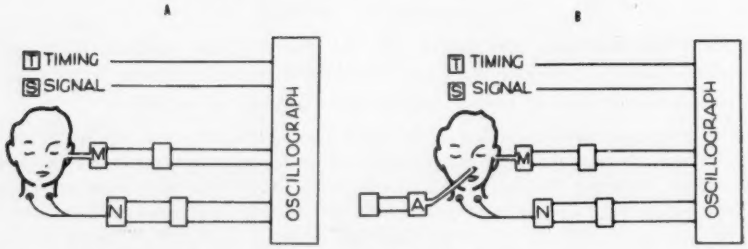

Fig. 1.—Experimental setup for eustachian tube studies.

A, arrangement for recording the nature of the change in transmission of "hum" sounds to the external canal with the eustachian tube closed and voluntarily opened. *M* is the microphone connected by tubing into the external canal. *N* represents the input to the amplifier from electrodes in the neck. Voluntarily produced "clicks" without swallowing and the sounds associated with swallowing are also recorded in this manner on either the loop oscillograph or the cathode ray oscilloscope.

B, similar arrangement with a sound source (*A*) for delivery to the nose. This sound picked up by the ear microphone (*M*) changes abruptly in intensity when the tube opens for a fraction of a second during swallowing. The duration of the swallowing act is timed from neck muscle activity recorded from electrodes *N*. *T* is a timing wave, usually 20 cycles per second, impressed on the record for measurement. *S* represents a hand signal sometimes used by the subject to place the time of some action.

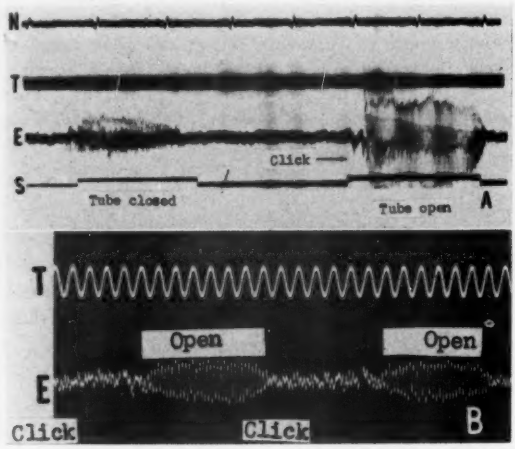

Fig. 2.—*A*, microphone in the external canal (*E*) records the increased amplitude of sound in the ear when there is humming with tube voluntarily opened as compared with when the tube is closed. *N* is the neck electrodes, which indicate no muscle activity, only pulse beat; *T* is a 60 cycle timing wave, and *S* is a signal operated by the subject to note the period of "hum" with open and closed tube. An arrow points to an acoustic transient heard as a click which was produced by the subject when voluntarily opening his tube.

B, a 100 cycle per second tone from the nose can be transmitted into the ear by voluntary opening of the tube. See ear microphone record (*E*). Just preceding each 100 cycle per second acoustic envelop, a strong negative pressure transient is recorded (arrows) when the sensitivity of the microphone is increased. This acoustic transient probably corresponds to the click heard by the subject when he voluntarily opens the tube. A clearer picture of this "click" is given in figure 4. *T* is a 20 cycle per second timing wave.

FINDINGS

The acoustic nature of autophony is illustrated in figure 2. The initial acoustic envelop of the sounds reaching the external canal when humming is partially the result of bone conduction agitation of the air in the external canal and air conduction from the nose through the air around to the tubing sealed in the external ear of the subject and sometimes leads to cancellation effects. The acoustic envelop of the sound reaching the external canal when humming with the tube voluntarily open indicates that there is an abrupt onset of the sound pattern, that the initial amplitude is more than double that of the first phonation, that the amplitude may fall off progressively. Sound in the head moving directly from the nasopharynx into the

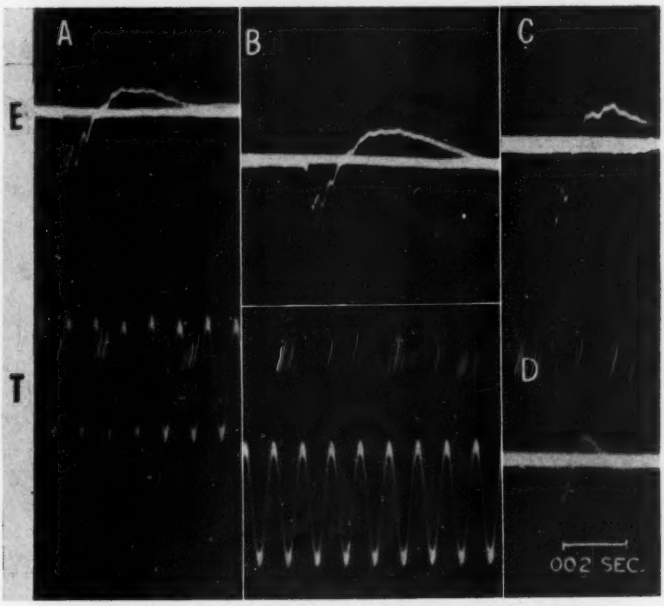

Fig. 3.—The click associated with voluntary opening of the tube can be detected by a microphone in the ear. (*E*) The poor low frequency response of the microphone preamplifier and the resonances in the tubing coupling the microphone to the ear interfere with an accurate recording of this voluntarily produced acoustic transient. The initial rapid drop (0.4 millisecond) of this transient is evident when compared with the 500 cycle per second timing wave. The total duration of the negative pressure is about 2 milliseconds. The rapid oscillations superimposed on the ascending limb of the negative pressure wave are in part due to tube resonances, while the immediately following prolonged positive pressure wave is related to the response characteristics of the microphone preamplifier.

T is a 500 cycle per second timing wave.

D, a transient produced by sudden separation of the moist lips.

middle ear through the opened eustachian tube moves the ossicular chain and drum membrane to initiate acoustic oscillation of air molecules in the external canal. Although sound production is carried out with uniform intensity of effort, as proved by initial monitoring of the acoustic envelop on the scope, there is an abrupt

increase of up to 12 decibels in the sound pressure recorded at the ear when the tube is voluntarily opened while humming. An initial sound pressure of about 80 decibels is recorded by the ear microphone when humming with the tube closed. An acoustic transient just preceding the voluntary opening is often recorded by the ear microphone and probably corresponds to a "click" heard by the subject. The

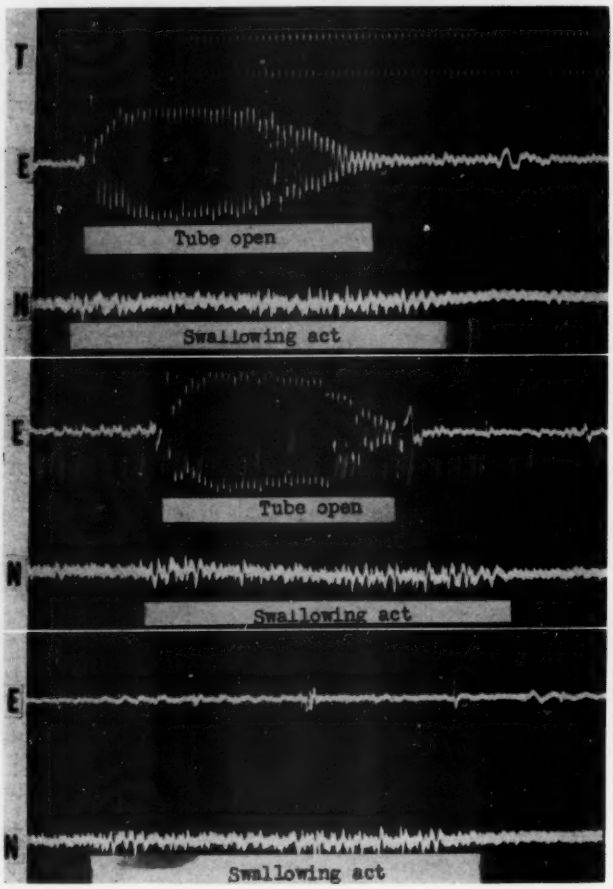

Fig. 4.—The duration and nature of the opening of the tube to transmit a 100 cycle per second tone from the nose into the middle ear. (Compare this with the 100 cycle per second timing wave *T*). Note the relation of tube opening in ear microphone record *E* to the swallowing act recorded as muscle action potentials from neck electrodes (*N*). The background activity from the ear microphone in bottom figure when the tube does not open indicates the acoustic insulation of the middle ear, obtained by the normal closed state of the tube, from the same 100 cycle per second tone introduced at the nose.

exact source of this transient is difficult to ascertain; it may be muscular, or it may result from sudden separation of opposed mucous surfaces in the tube. The conduct-

ing mechanism and the drum membrane react to this transient pulse and, in turn, initiate the pulse wave moving in the air molecules to the diaphragm of the ear microphone.

Yule [8] described the click produced by opening of the tube. He stated:

It is a sharp crackling sound which is referred to some part of the tympanum perhaps to the membrane itself. This I have no doubt whatever is caused by the separation of the walls of

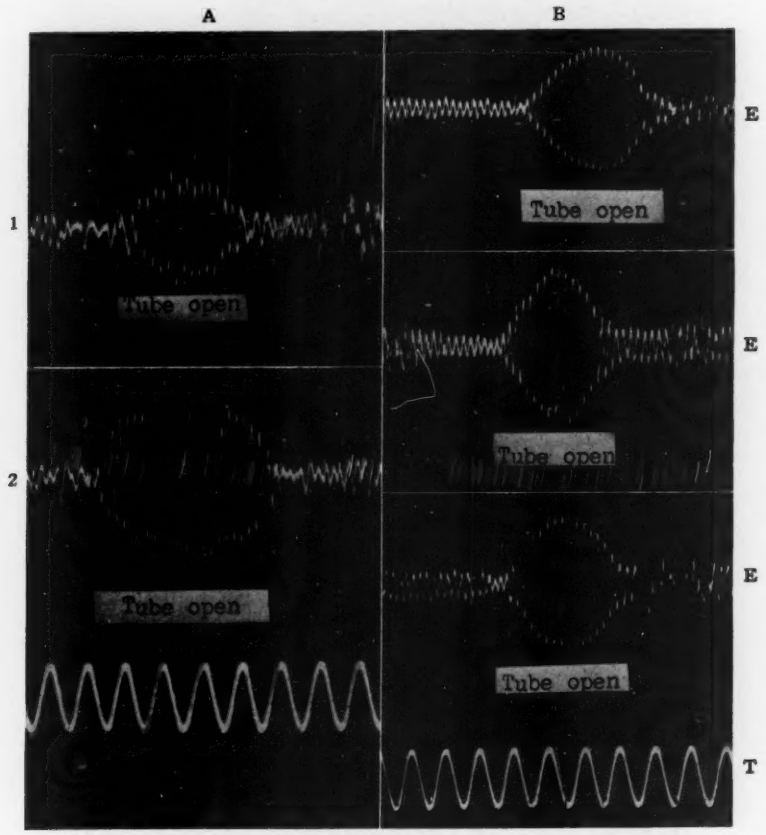

Fig. 5.—The reflex opening of the tube during swallowing varies with conditions. In *A 1* the subject swallowed his saliva on command; in *A 2* the subject swallowed a little water on command. The latter produced a greater and longer opening of the tube, as indicated by the length and height of the 100 cycle per second acoustic envelop recorded by the ear microphone (*E*). In general the degree and duration of tubal opening under uniform conditions may be rather constant for a given subject. Note similarity of the acoustic envelops in all three records of the same subject (right side). While there are slight variations they are not so great as the variations between different subjects (fig. 5 *B*).

T is a 20 cycle per second timing wave.

8. Yule, C. T. F.: On the Mechanism of Opening and Closing the Eustachian Tube, J. Anat. & Physiol. **8**:127-132, 1873.

the Eustachian tube. The sound can be heard by anybody at the commencement of the act of swallowing and forms the prelude to a series of noises arising from the moving mucus, etc., about the posterior nares and pharynx.

Bekesy [9] recorded this click and from its shape and duration obtained evidence about the resonance frequency and damping of the conducting mechanism that was

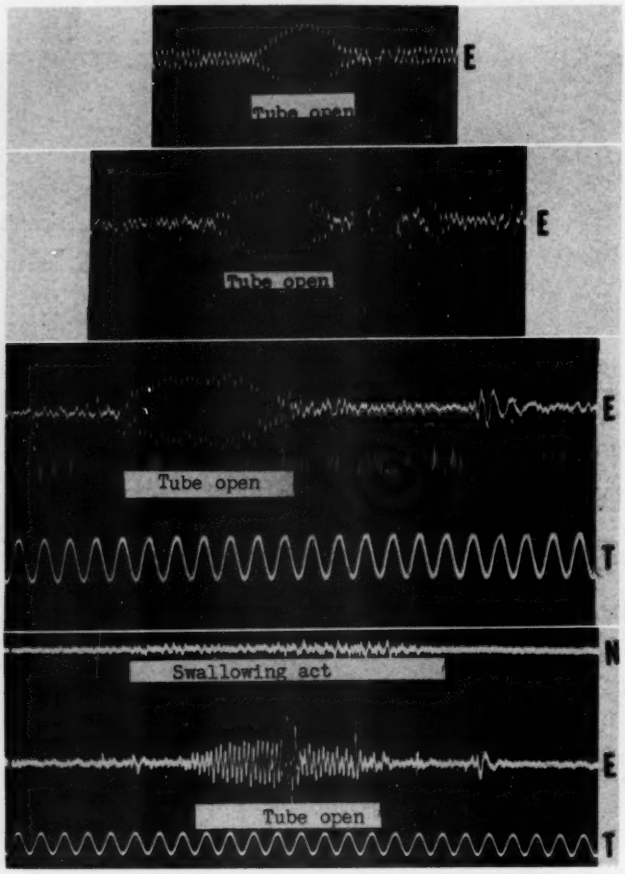

Fig. 6.—Note the differences in the duration and type of tubal opening between subjects. Each record is from a different subject performing under similar conditions. Note the short opening in the top record as compared with the lower record. The length of the label under the 100 cycle acoustic envelop indicates the duration of tubal opening.

The fourth record shows the relation of tubal opening to the act of swallowing. The 100 cycle transmission during tubal opening is seen in the ear microphone records (*E*). The muscle action during swallowing is recorded at *N*.

T is a 20 cycle per second timing wave.

9. Bekesy, G.: Über die mechanisch-akustischen Vorgänge beim Hören, Acta oto-laryng. **27**:281-296, 1939.

set into motion by this transient. In our experiments similar transients were produced voluntarily and were observed in the record of voluntary opening of the tube to a sound delivered at the nose. The length of the tubing coupling the microphone with the canal affects the recorded response of the transient because of resonances.

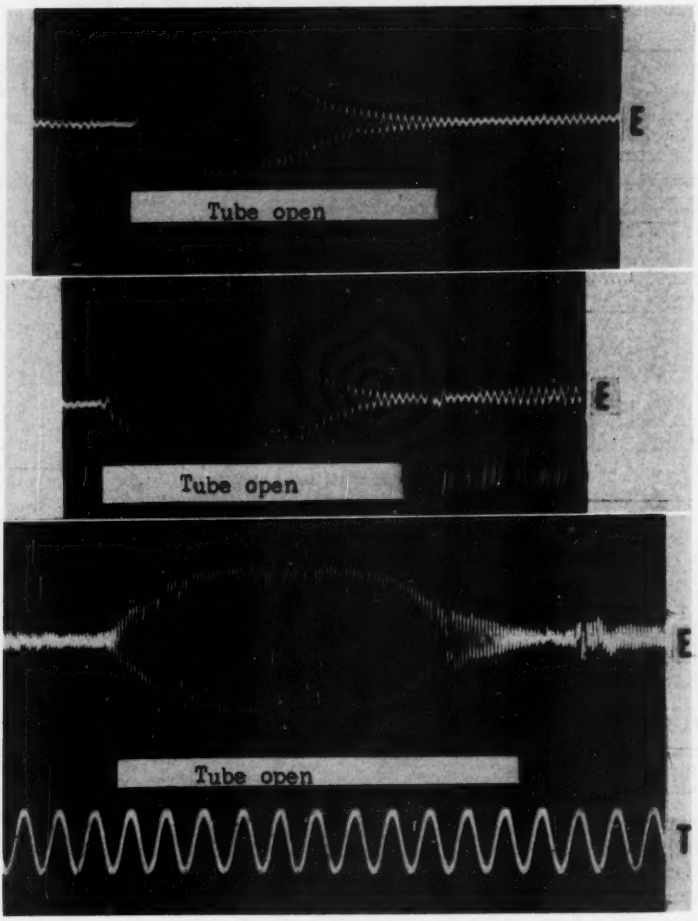

Fig. 7.—With a uniform position of the same subject, the swallowing of a little water each time produced some variation in the degree and duration of tubal opening as expressed in the different acoustic envelopes of the transmitted sound recorded by the ear microphone (*E*). *T* is a 20 cycle per second timing wave.

This was investigated by changing this tube length. The frequency of the small waves superimposed on the upward slope of the initial wave was greater with shorter tubing. The duration of the first major wave was 2 milliseconds with an initial rise of about 0.4 millisecond. The prolonged weaker positive wave, lasting

7 milliseconds, probably resulted from a poor response of the microphone preamplifier.

The direction of this major pressure pulse is negative, suggesting that it may be produced by an inward movement of the drum. This could be explained by the separation of the walls of the tube and abrupt breaking of the mucous blanket. This suddenly increases the active volume of the air behind the drum, thus reducing its pressure and causing an inward motion of the drum. The slower return of the pressure wave to the zero position suggests that this is followed by a slower movement of the drum back to its resting position that is in large part determined by the resonance frequency of the drum with its attached conduction mechanism.

Duration (in Seconds) of Tubal Opening and Swallowing

Subject	Period 1	Period 2	Period 3
Reflex Opening of the Eustachian Tube			
1	0.17	0.16	0.13
	0.16	0.15	0.17
	....	....	0.19
2	0.21	0.20	....
	0.10	0.15	....
3	0.43	0.39	0.24
	0.60	0.35	0.25
	0.45	0.39	0.30
	0.23	0.39	0.31
	0.40	....	0.40
	....	....	0.39
	....	....	0.32
	....	....	0.30
Voluntary Opening of the Eustachian Tube			
3	0.70	0.27	0.55
	0.52	0.35	0.45
	....	0.35	0.55
Duration of Swallowing Potentials			
1	....	....	1.00
	....	....	0.9
	....	....	0.95
	....	....	1.00
2	....	....	0.8
	....	....	0.6
	....	....	0.8
	....	....	0.6
3	....	....	1.0
	....	....	1.0
	....	....	1.0
	....	....	1.0

Discounting the distortion in the record due to the tubing and the microphone preamplifier, the induced oscillation of the drum appears to stop abruptly, indicating a high degree of damping of the conducting mechanism. The whole cycle of movement is heard as a "click" (fig. 3). Abrupt separation of the moist lips produces a similar transient, with an initial negative wave lasting about one thousandth of a second (fig. 3 *D*). This suggests that breaking of a mucous film across the lumen of the tube may be the source of the "click" heard and recorded with tubal activity.

The initial negative pressure had a peak value of about 70 decibels.

The presence, degree and duration of tube opening during swallowing are evidenced by sound transmission.

The tube sometimes opens abruptly and closes more gradually. The conditions of the recording equipment are set so that the 100 cycle background does not appear in the ear microphone pick-up during the closed state of the tube. Therefore

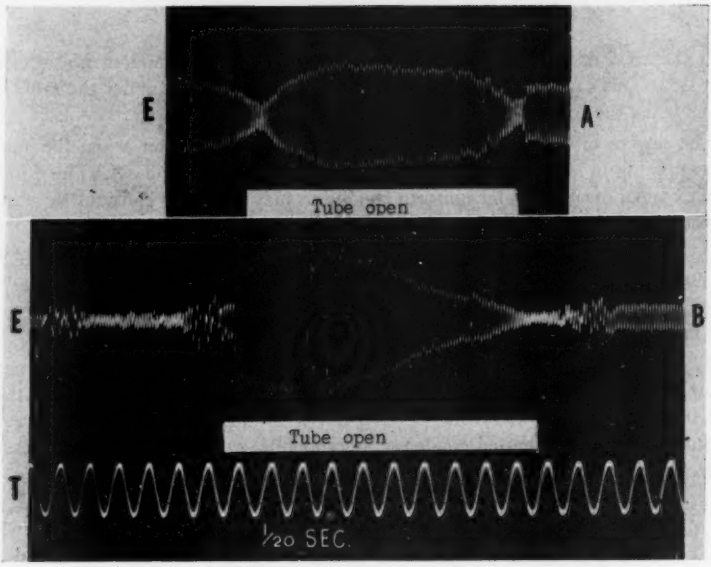

Fig. 8.—The difference in the degree and duration of the voluntary opening of the tube (*A*) as compared with reflex opening during swallowing (*B*) is shown. The tube is seen to open more abruptly and more widely but to close more gradually during the reflex action than during voluntary action.

T is a 20 cycle per second timing wave.

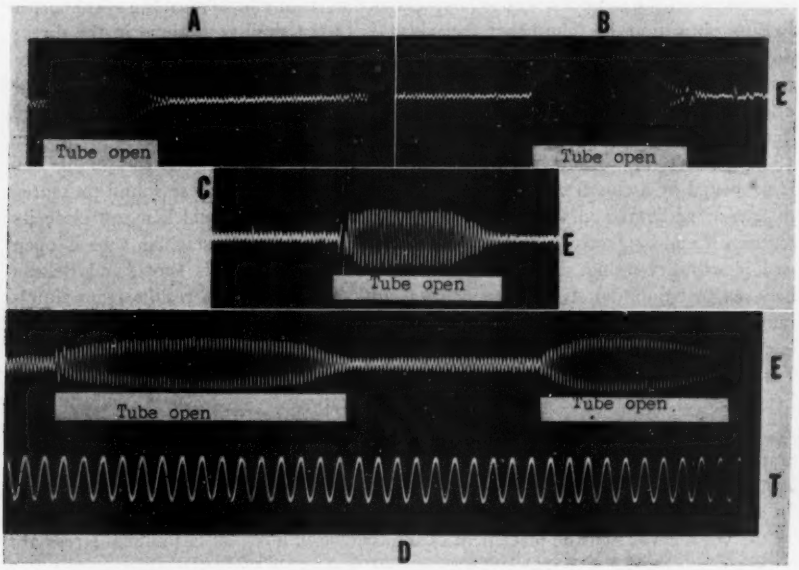

Fig. 9.—The differences between voluntary and reflex opening of the tube recorded by the ear microphone (*E*) is shown. In *A* the tube is voluntarily opened; in *B* the tube opens reflexively during swallowing. Note the difference in the amplitude and duration of the transmitted 100 cycle per second tone envelops. In *C* the tube opens reflexively during swallowing as compared with *D* in which it is voluntarily opened.

T is a 20 cycle per second timing wave.

when the tube does not open during swallowing, only the random noise will be recorded. This can be easily compared with the records made under the same conditions while the tube opens (fig. 4).

The degree of reflex tubal opening may vary, being greater when the subject has something in his mouth to swallow besides the saliva (fig. 5 *A*). The duration of reflex tubal opening under uniform conditions may be rather constant for a given subject (fig. 5 *B*) and may be quite unlike that of another subject (fig. 6). However, slight differences in tubal opening are seen in the same subject in successive swallows (fig. 7). The tube opens for a short period during the swallowing act, and the opening begins shortly after the muscle activity starts (fig. 4).

The values of some of these time intervals are recorded in the table.

The tube opens more widely during the swallowing reflex than during voluntary opening (figs. 8 and 9). An increase of sound pressure of 20.6 decibels with the tube opening during swallowing to transmit a sound delivered at the nose was measured with the calibrated condenser microphone, as compared to an increase of 12 decibels during voluntary opening for transmission of the same sound.

COMMENT

The understanding of the nature of sound transmission through the head during phonation has been largely clarified by the investigations of Bekesy.[10] His findings afford a proper perspective for the interpretation of the acoustic phenomena related to tubal function as described in this report. Bekesy points out that the hearing of one's own voice normally is due in part to bone conduction and in about an equal part to air conduction. The sound pressure produced in the mouth cavity contributes about the same amount of loudness as that from vibration of the vocal cords. During phonation, he found that the vibration of the skull is minimal in the direction parallel to the axis of the ear canal and maximal in the vertical direction. He also found that there is an average drop of about 20 to 25 decibels in the sound intensity of speech at the lips to the opening of the ear. On the other hand, he found that the sound pressure in the mouth 8 cm. behind the lips is much higher (30 decibels) than 3 cm. in front of the lips. Now while in man the middle ear is protected by a closed eustachian tube from direct access of these sound pressures of speech generated in the air cavities of the head and the chicken can close its external canal to protect the ear while crowing, the frog's eustachian tube is open widely while croaking. However the frog's eustachian tube is short, and Bekesy believes that minimal stimulation of its conducting mechanism results from simultaneous, in phase, sound pressure to the two sides of the drum membrane. The body is confronted with the problem of producing sound within it for projection in space (as in speech) without unduly disturbing at the same time the sense organ in the head used to detect these air-borne acoustic vibrations.

Bekesy has considered some of these acoustic vibrations generated within and transmitted through the head and the ways in which the embedded recording apparatus of the ear is naturally protected from these stimuli. He points out that, in addition to closure of the eustachian tube in man, the symmetrical movements of the vocal cords and the placement of the cochlea in a region of minimal stress are

10. Bekesy, G.: The Structure of the Middle Ear and Hearing One's Own Voice by Bone Conduction, J. Acoustical Soc. America **21**:217-232 (May) 1949.

important. The ossicles are so suspended and arranged that vibrations of the skull are not easily transmitted to them. The skull itself was found to vibrate as a rigid body below 1500 cycles per second. In the sound "oo" the vertex vibrated more, the forehead less and the mastoid bone practically none. There is a large vertical component in the vibrations of the skull in phonation. This probably resulted from the vertical vibration of the edge of the cords and the acoustic vibrations within the mouth. This does not produce much lateral vibration of the skull because of the lateral symmetry of the head. Mullendore [11] also found that during vowel production the vibrations of the mastoid were much smaller than those of the vertex. Bekesy found that when the position of the head is symmetrical to the body, the phonation of "z" produces the same amplitude in the vibration pick-ups from the two temporal bones. Tilting the head to one side reduces the vibration amplitude on the side to which the head is tilted and increases it on the opposite side. The difference is about 10 to 20 per cent of the total vertical movement. Even walking produces a transient head vibration, lasting about 1/200 second.

According to Bekesy, hearing by bone conduction can originate in the middle ear in two ways: 1. When the head is moved, the inertia of the ossicles produces a movement of the ossicles relative to the cochlea. 2. A motion transmitted to the skull deforms it in such a way that a relative movement is produced between the ossicles and the cochlea. He points out that the middle ear is placed in a zone not subject to tension. The hammer and incus are built so that the center of gravity and the point of rotation are the same. With the center of rotation near the edge of the drum membrane, a head is required on the malleus to bring the center of gravity to this point. If the skull vibrates parallel to the axis of the ear canal, then the mass of the malleus head acts to rotate the hammer in a direction opposite to that of the mass of the handle. Therefore the movements caused by inertia forces of the handle are compensated for by the head of the malleus. Observations on a solid ring subjected to stress indicate that when a stress is applied to the skull, large deformations in the direction of the ear canal (perpendicular to the bony wall) are not expected. During phonation the middle ear seems to be well protected against vibration and deformation parallel to the axis of the ear canal. On the other hand, this kind of protection is not important, since during phonation the vibration of the skull is minimal in the direction parallel to the axis of the ear canal and maximal in the vertical direction. Bekesy shows further that the middle ear is built in such a way that hearing by bone conduction caused by vertical vibration is also minimized. A displacement of the hammer in the direction of its long axis does not move the labyrinth fluid; the stapes rotates around its long axis, and fluid moves only from the upper and lower edge of the footplate.

Since the head vibrates largely in a vertical direction during phonation, the long axis of the footplate is at right angles to the vertical axis of the neck. On the other hand, Bekesy points out that in the frog and chicken the ossicles are not balanced, the hammer does not have a compensating head and the stapes footplate is round, not elliptical.

He observes further that if the human ossicles are moved in a horizontal plane at right angles to the axis of the ear canal no fluid displacement is produced in the

11. Mullendore, J. M.: An Experimental Study of the Vibration of the Bone of the Head and Chest During Sustained Vowel Sounds, Speech Monogr. **16**:163-176 (Sept.) 1949.

cochlea, because the joint between the stapes and the incus is easily moved. The greater stiffness of the annular ligament over other elastic parts of the ear allows compression of the middle ear and movement of the ossicles relative to the wall without cochlear fluid movement.

It is necessary to maintain constant recording conditions when studying changes in acoustic transmission from the nose into the external canal. Opening of the mouth, a poor seal at the nose and other factors influence the response of the microphone connected by tubing to the external canal.

The controlled quantitative measures in this report represent real major changes in the acoustic pathway from the nose to the external canal and can be explained only by opening of the closed tube between the two regions. The sound wave pattern is an expression of such details of the tube opening as duration, degree and manner of opening and closing.

Using this index of tubal function, one can see that the tube does not open during phonation and often fails to open during swallowing. As shown in the previous report, this failure to open can be explained sometimes by the associated intact mucous blanket, bridging the visible area of the tube mouth. One sees that the tube can be opened voluntarily, but this type of opening differs from that produced reflexly, being less wide. This seems reasonable, since the reflex contraction of the tubal muscles associated with swallowing may be more vigorous than the voluntary contraction. Also the duration of reflex opening is shorter than that of voluntary opening of the tube. One notes further that the tube opening varies, even when it occurs reflexly during swallowing (depending on such a factor as whether one swallows only saliva or water in addition). Variations in the degree and duration of reflex contractions of the tubal and palate muscles may vary depending on the amount of material in the mouth when swallowing. It is indeed often difficult to complete the swallowing act on command when no extraneous matter is present in the mouth, and only incomplete initial movements of the palate, etc., result. These can be seen in the moving picture records described in the previous report. With use of similar amounts of the same substance (water), the degree, duration and type of tubal opening is seen to vary with individuals. This seems reasonable, since the musculature and physical dimensions of this region vary from person to person and the act of swallowing produces different muscle forces on the tube in different persons.

The pattern of opening appeared to be characteristic for a given individual. This is also compatible with the idea that in any given individual the pattern of tubal opening is determined by the special anatomical conditions such as the size of the muscles, the amount of fat and glands adjacent to the tube and the condition of Rosenmüller's fossa. Very slight variations can still be made out from one reflex opening to another in the same subject while the general pattern remains the same. That the tubal opening may vary in pathological states seems evident, and theoretically one might imagine that the tube may open so poorly that only one cycle of a 100 cycle sound wave is transmitted. The opening of the tube may be related to the breaking of the mucous blanket. A mucous film may break in less than a millisecond, but how rapidly it may re-form is not known. The analysis of the click record suggests that the initial negative wave, which reaches a maximum in about 0.4 millisecond, may be the result of breaking of this mucous film.

Analysis of the click record also gives evidence to corroborate other observations indicating that the conducting mechanism is highly damped and that its resonance frequency is somewhere between 500 and 1000 cycles per second.

PHYSIOLOGIC PRESSURE CHANGES IN THE NASOPHARYNX

The eustachian tube and middle ear are exposed physiologically not only to sound pressures in the nasopharynx—during phonation—but also to slower pressures arising from respiration and deglutition. The pressure changes taking place at the mouth of the tube during quiet respiration are small, and there is no associated activity of the tubal muscles at this time. The eustachian tube remains closed to shield the middle ear from them. Even the pressure changes in the nasopharynx during forced respiration are not sufficient to open the tube. Ordinarily during swallowing the open state of the nose does not allow any pressure changes to develop opposite the mouth of the tube, but under conditions of nasal obstruction one may generate definite pressure changes in the nasopharynx comparable to those produced by Toynbee's maneuver (swallowing with mouth and nose closed). These pressures are created at the mouth of the tube just when the tubal muscles are actively contracting, tending to open the tube, and hence such pressures may be transmitted to the middle ear. To measure them at the nose two methods were used. One consisted in electrically recording the movements of a tight rubber diaphragm by means of a special capacitative probe. Amplifier and galvanometers were used with optical recording on film. The diaphragm closed a small air cavity connected by tubing to the nose. With a glass nasal tip fitted into one nostril, this cavity became part of the air system of the nose and nasopharynx, and, with the other nostril closed, pressure changes within this air system affected the diaphragm. The volume of air in the tubing and behind the diaphragm was relatively small compared with that in the nose, sinuses and nasopharynx and hence did not contribute much distortion of the pressure changes within the nose and elsewhere. The resonance frequency of the diaphragm was set well beyond the periods of the pressure waves, so that the diaphragm would follow the pressure pattern rather well. The damping of the tense rubber diaphragm was sufficient to prevent much after-vibration.

The other method of recording these pressures consisted in the use of a tambour driving a marker on a smoked paper kymograph. The resonance frequency of the tambour diaphragm was higher than that of the pressure waves. The damping was not sufficient to prevent a little rapid after-vibration. A calibration was carried out on both types of recording systems to allow measurement of the duration, type and quantity of the pressure changes developed when the patient was swallowing with the nose closed. The records obtained show that rapid pressure changes with both the positive and the negative phase develop in the nose and nasopharynx during swallowing with the nose closed (fig. 10). The pressure changes vary in degree and type from subject to subject and in each subject in different swallowing acts. The duration of the pressure changes is about 0.8 second, and the amount of positive pressure varies but is about 10 cm. of water, while the maximum negative pressure is also 10 cm. of water. The creation of these small pressure changes in Toynbee's maneuver afford a convenient test for the adequacy of tubal function during swallowing. If the tube can open and transmit these small

pressures, it cannot be badly obstructed. Evidence for a positive Toynbee test is obtained by observing the drum movement. The two types of drum movement considered significant are a sucking in of the drum and a sucking in followed immediately by an outward movement returning the drum to its normal position. This indicates that the tube does not open during the initial period of positive pressure. The varying nature and duration of the pressure changes recorded from the nose in Toynbee's maneuver explain why there are variations in the observed drum movement. The exact cause for the pressure changes developed in the nose and nasopharynx while swallowing with the nose closed is not known. The nasopharynx is hermetically sealed from the pharynx in swallowing by the contracting soft palate. With the nose closed, a closed air cavity is produced with only one actively changing wall (that bordered by the soft palate). As the palate rises to close the naso-

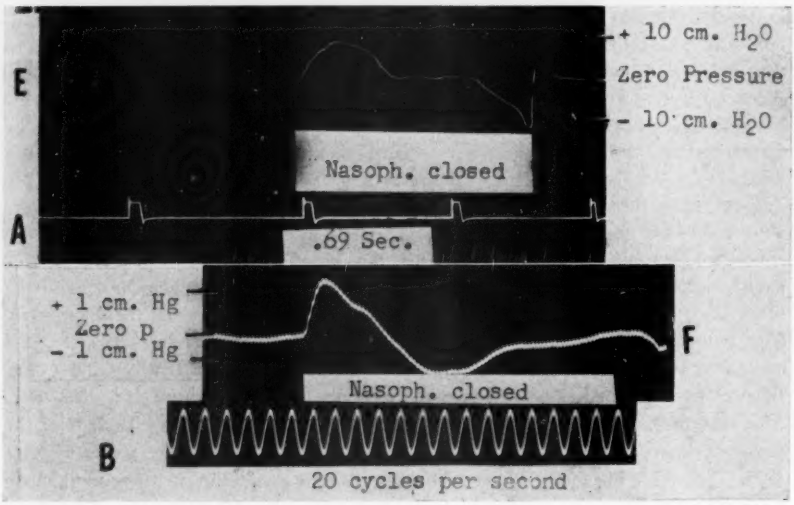

Fig. 10.—*A*, record of the pressure changes in the nose and nasopharynx during Toynbee's maneuver (made on a smoked paper with a tambour). The degree and duration of the pressure changes vary. A maximum positive or negative pressure of about 10 cm. of water was recorded. The maneuver creates an initial positive pressure which is followed by a negative pressure wave of about the same intensity but different shape. A short period of zero pressure may separate these.

The interval between two time marks in *T* is about 0.69 seconds.

B, records of the pressure changes in the closed nasal chambers when the soft palate closes off the oropharynx during swallowing (Toynbee's maneuver). An initial positive pressure is followed by a longer negative pressure. The degree and duration of these pressure changes vary. A maximum pressure of about 1 cm. of mercury is recorded. The tube is probably not open during the initial period of positive pressure but opens during the following negative pressure, as reflected in the movements of the drum. A possible explanation of these pressure changes is given in the text.

The timing wave is 20 cycles per second.

Note that the capacitative probe method for electrical recording of these pressures gave wider galvanometer movement for positive than for the same amount of negative pressure.

pharynx, not only the oropharynx and hypopharynx enlarge but the cricopharyngeus muscle relaxes to permit continuity between these cavities and the esophagus.

A sudden enlargement of the space occupied by the gas in the throat results in a reduction of the pressure of the air in this region. The pressure on the under side of the soft palate is thus reduced with respect to the air above the soft palate, and the soft palate is momentarily pushed back into the oropharynx without breaking the seal between the two cavities. This downward movement increases the volume of the air in the closed spaces above and reduces its pressure. The duration of this negative pressure change may be related to the duration of opening of the cricoid sphincter. A preliminary positive pressure recorded in the nose with Toynbee's maneuver may be due to a bulging up of the soft palate after initial closure of the nasopharynx from the pharynx with an increase of pressure in the oropharynx before the cricoid sphincter opens. Révész [12] observed peak pressures of 130 mm. Hg in the oropharynx during swallowing. The momentary reduced volume of the upper closed air system between the nasopharynx and the closed nostrils results in a small increase in its pressure. This may indicate that closure of the nasopharynx is synchronized neurologically so that it immediately precedes opening of the cricoid sphincter. The initial rise of the palate followed by a fall can be observed during swallowing in the subjects with defects of the face. If one makes an inspiration effort with the mouth and nose closed just before swallowing, a greater degree of pressure change is developed in the nasopharynx as the muscles of the tube move to separate the walls of the tube. Direct observation of the soft palate and tube during swallowing reveals differences in movement from that in speech, but these pressure changes recorded above cannot exactly be predicted by observing the palate motion.

SUMMARY

An experimental procedure is described that permits quantitative observations on the nature of the physiological opening of the eustachian tube.

Direct evidence of the acoustic nature and degree of sound pressure changes in "autophonie" is obtained.

Acoustic transients are recorded that are associated with the swallowing act and the voluntary opening of the tube.

Palate and tubal activity as produced in phonation is not associated with opening of the tube.

Swallowing water is more effective than swallowing without water in opening the tube.

The tube is often closed even during swallowing. The reflex opening of the tube during swallowing is different from the voluntary opening. In the former the tube is more widely open. Other differences noted are in duration and rate of opening and closing.

The degree, type and duration of the pressure changes within the nose and nasopharynx in Toynbee's maneuver are recorded, and an explanation is suggested for them.

The assistance of Mr. J. E. Hind in the development and use of the electronic equipment used in these studies is acknowledged.

12. Révész, G.: Die Rolle von intraoralen Druckfactoren in der Pathologie der Tonsillen. Pract. oto-rhino-laryng. **11**:193-205, 1949.

TREATMENT OF PULMONARY COMPLICATIONS IN NEUROSURGICAL PATIENTS BY TRACHEOTOMY

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SINCE the advent of modern surgery, prevention and treatment of postoperative pulmonary complications have been subjects of intensive interest and research. In 1924 Leopold [1] first pointed out the similarity of physical findings in postoperative atelectasis and in instances of foreign bodies obstructing a bronchus. This led to the original work of Lee and Tucker,[2] who successfully treated postoperative atelectasis by bronchoscopy. In attempting to reproduce the condition experimentally, Tucker, Lee and Clerf [3] inserted secretions which had been previously aspirated from a patient into one of the main bronchi of dogs. They found that atelectasis could be produced by this method, but only after the animal was anesthetized deeply enough to abolish the cough reflex. From these experiments was developed the present concept that two factors are responsible for postoperative atelectasis: (1) suppressed or ineffectual cough and (2) presence of abnormal secretion in the tracheobronchial tree. Management of this problem has been discussed by many observers,[4] and specific therapy is always directed toward relief of mechanical obstruction.

Patients subjected to abdominal and even thoracic surgery usually recover an effective cough within a few days. Following intracranial surgery, however, a diminished state of consciousness occasionally persists many days, with suppressed cough and stagnation of secretions. Constant vigilance is necessary to prevent atelectasis with the resulting pneumonitis and consolidation. Scrupulous care, including frequent turning of the patient, tracheal aspiration, and hyperventilation by means of blow bottles or carbon dioxide inhalations, has diminished the incidence

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1. Leopold, S. S.: Post-Operative Massive Pulmonary Collapse and Drowned Lung, Am. J. M. Sc. **167**:421-433, 1924.

2. Lee, W. E., and Tucker, G.: Post-Operative Pulmonary Complications, Tr. Coll. Phys. Phil. **47**:231-245, 1925.

3. Lee, W. E.; Tucker, G., and Clerf, L. H.: Post-Operative Pulmonary Atelectasis, Ann. Surg. **88**:6-14 (July) 1928.

4. Dripps, R. D., and Deming, M. V.: Post-Operative Atelectasis and Pneumonia: Diagnosis, Etiology and Management Based upon 1,240 Cases of Upper Abdominal Surgery, Ann. Surg. **124**:94-110 (July) 1946. Haight, C., and Ransom, H. K.: Observations on Post-Operative Prevention and Treatment of Atelectasis and Bronchopneumonia, ibid. **114**:243-262 (Aug.) 1941.

of such complications. Antibiotics are used systemically to combat infection which always follows mechanical obstruction. Nevertheless, an appreciable number of obstructive pulmonary complications occur and constitute one of the most formidable postoperative complications of neurosurgery.

Certain features about patients following intracranial surgery make the usual methods of clearing the lungs impractical: (1) depressed cough over an extended period, (2) aspiration of food or liquids into the lungs (with or without weakness of the ninth and tenth cranial nerves), (3) obstruction by relaxed pharyngeal musculature and (4) reaccumulation of obstructive secretions after bronchoscopy or tracheal aspiration. We have resorted to tracheotomy in certain selected cases as the best method of keeping the lungs constantly clear and enhancing oxygenation. Other patients presented obstructive symptoms which demanded tracheotomy, as in laryngeal paralysis, spasm, or edema following endotracheal anesthesia.

This report presents the cases of 15 neurosurgical patients after operation who were aided by tracheotomy.

REPORT OF CASES

CASE 1.—W. H., a male Negro infant aged 14 months, had a third ventricle colloid cyst removed by suboccipital craniectomy on Jan. 27, 1948. The child did well postoperatively, although requiring frequent pharyngeal aspiration. Nutrition was supported by interval gavage feedings. During the next two weeks, laryngospasm tended to develop with each aspiration or feeding. There were also many spontaneous short episodes of spasm attributed to laryngeal stimulation by accumulated secretions. During one severe spasm, oxygen and repeated aspiration were necessary to relieve cyanosis. On February 6 tracheotomy was performed by employment of the technic described by Tucker,[5] with the bronchoscope in the trachea during the procedure. At this time the larynx appeared normal, and after tracheotomy there were no further anoxic episodes.

The patient continued to have increased intracranial pressure and died 41 days after tracheotomy. At autopsy the lungs were normal.

Comment.—Laryngospasm presented no problem with an airway guaranteed by tracheotomy.

CASE 2.—G. P., a white man aged 42, had a temporal glioma partially removed by craniotomy April 20, 1948. Postoperatively he was severely depressed, with absence of cough reflex and profuse pharyngeal secretions which had to be aspirated at least every hour. Five days after operation roentgen and physical signs of atelectasis of the lower lobe of the right lung appeared. By bronchoscopy considerable thick, purulent secretion was removed and aeration temporarily improved. Because atelectasis soon reappeared, bronchoscopy was repeated daily, with interim catheter aspiration of trachea. Even during bronchoscopy the cough was ineffective and atelectasis was not completely relieved. Tracheotomy was performed May 2, following which effective aspiration markedly improved pulmonary ventilation and no clinical signs of anoxia were evidenced.

Pressure from the malignant growth and pulmonary infection caused death on May 7.

Comment.—Absence of the cough reflex due to the depressed condition permitted accumulation of secretion in the smaller bronchi which aspiration through the tracheotomy tube could not reach.

CASE 3.—H. M., a white man aged 73, was admitted May 28, 1948 in a somnolent, almost comatose, condition. On June 2, before a definite diagnosis could be reached, complete atelectasis of the left lung and typical signs of laryngeal obstruction developed. Direct laryngoscopy

5. Tucker, G.: Technique of Tracheotomy with Use of Sulfonamides in Prevention of Wound Infection, Tr. Am. Laryng. A. **64**:186-190, 1942.

revealed adduction of both cords with exudate, crusting and granular hyperemic mucosa of pharynx and larynx. Introduction of a bronchoscope relieved the laryngeal dyspnea. Profuse secretion was aspirated and contained elements similar to gastrostomy feedings, indicating regurgitation with aspiration of vomitus into the lungs. With the bronchoscope remaining in position, tracheotomy was performed.

The patient was much improved, with good color and quiet respiration. Atelectasis was completely relieved, as shown by chest roentgenograms before and after tracheotomy. On June 5, a large subdural hematoma was evacuated through burr holes. Six weeks after operation the larynx appeared normal, and decannulation was accomplished by decreasing the size of the tracheotomy tube, then occluding it with a cork for three successive days and nights.

He was discharged in good condition and has remained well.

Comment.—Tracheotomy, by solving the double problem of laryngeal obstruction and pulmonary atelectasis, kept the patient alive long enough for specific neurosurgical therapy to effect a cure.

CASE 4.—R. T., a white woman aged 61, had a parieto-occipital glioma partially removed by craniotomy July 1, 1948. Postoperatively the patient was comatose, and on July 6 labored respiration with stridor and cyanosis developed. Direct laryngoscopy revealed both cords fixed near the midline, with slight adduction during inspiration. A bronchoscope was inserted, promptly relieving dyspnea, and tracheotomy performed. Within a few hours the patient became more responsive, progressively improving so that in five weeks she was able to leave the hospital. The larynx had returned to normal, and the tracheotomy was decannulated. Recurrence of the tumor caused death three months later.

Comment.—Laryngeal obstruction may have been a function of cerebral irritation and resulting spasm. Rapid response after tracheotomy indicates the previous five days of coma might have been partially due to unrecognized anoxia.

CASE 5.—F. P., a white man aged 70, was struck by a trolley July 22, 1948. During the next few hours he became gradually more somnolent, with evidence of rising intracranial pressure. Burr holes revealed no subdural bleeding, and the diagnosis was subarachnoid bleeding plus concussion and contusion. He remained comatose, with the airway a constant problem due to profuse secretion and relaxed pharyngeal musculature. Despite frequent catheter aspiration, right-sided atelectasis and pneumonia appeared (clinical and roentgen diagnosis). Daily bronchoscopy provided temporary improvement, but accumulation of secretion soon caused return of the collapse. Tracheotomy was performed August 3. By frequent aspiration and instillation of penicillin directly into the tracheotomy tube, as recommended by Tucker and Atkins,[6] the chest was kept completely clear. With this therapy there was gradual lightening of the stupor, but after about six weeks the cardiovascular system failed. On October 9, after more than two months in coma, he died of bilateral pneumonia, with cardiac failure and cerebral trauma as causes.

Comment.—Tracheotomy was performed to improve oxygenation by clearing pulmonary disease. The temporary improvement indicated that anoxia was at least partly responsible for the stupor.

CASE 6.—A. J., a Negro woman aged 58, had a large acoustic neuroma removed from the left side by suboccipital craniectomy Aug. 25, 1948 under endotracheal anesthesia. Postoperatively the patient was very depressed, with inability to swallow and accumulation of secretions. Intratracheal aspiration afforded some relief, and on two occasions a soft rubber endotracheal tube was inserted under direct vision to facilitate aspiration. Two days after operation signs of laryngeal obstruction appeared, with rales throughout both lungs and

6. Tucker, G., and Atkins, J. P.: Recent Trends in Bronchologic Use of Chemotherapeutic and Biotherapeutic Agents, Ann. Otol., Rhin. & Laryng. **53**:777-786 (Dec.) 1944.

atelectasis of the lower lobe of the left lung. Direct laryngoscopy showed the left cord fixed in midposition, with exudate over both cords and subglottic areas. With the bronchoscope in position, tracheotomy was performed. From this time the patient had a clear airway and quiet respirations. However, she never became completely responsive and died suddenly of a pulmonary embolus September 13.

Comment.—The trauma of tracheal aspiration and intubation were sufficient, with one vocal cord paralyzed by the tumor, to cause laryngeal obstruction.

CASE 8.—A. S., a white man aged 52, had a two stage sympathectomy performed for severe hypertension. During the second stage, on September 13, cardiac arrest occurred. Massage was begun, and the heart started beating spontaneously although a period of about four minutes had passed without any cardiac action. Postoperatively he remained comatose, and an endotracheal tube was kept in position to ensure the airway as well as simplify aspiration and oxygen administration. When unconsciousness had persisted 48 hours, tracheotomy was performed to protect the larynx from irritation of the endotracheal tube. The patient lived five weeks, although never recovering consciousness. At autopsy the lungs were normal, and death was attributed to irreversible cerebral anoxia and myocardial weakness.

Comment.—When endotracheal tubes remain in position for more than 12 hours, they often become partially or completely obstructed by crusting at the distal end. In this manner they may completely block the airway and should always be changed at least twice daily.

CASE 9.—M. M., a white woman aged 29, had an acoustic neuroma removed completely from the right side by suboccipital craniectomy on Jan. 21, 1949. There was severe postoperative depression, with inability to swallow, inactive cough reflex and accumulation of secretions in the pharynx and trachea. On January 24 physical signs and roentgenograms indicated atelectasis of the lower lobe of the left lung. Bronchoscopy confirmed this and also showed midline paralysis of the right vocal cord. With the bronchoscope remaining in position, tracheotomy was performed. The patient's general condition improved, but the swallowing function remained absent so that food, liquids or pharyngeal secretions were consistently aspirated into the lungs. For this reason the tracheotomy tube could not be safely removed, even though there was motility in the right cord on April 29 and the oral airway was adequate. Gavage feedings were required for almost five months, after which some swallowing function was recovered. When this occurred, tracheal aspiration was unnecessary and the tracheotomy was decannulated. Mirror examination revealed a normal larynx, and the patient was discharged July 22.

Comment.—Although the patient aspirated into her lungs almost everything she attempted to swallow, removal through the tracheotomy tube by a suction catheter prevented serious consequences.

CASE 10.—A. P., a white man aged 52, was admitted Feb. 1, 1949 with a diagnosis of meningitis and brain abscess. Because of poor respiratory action, an endotracheal tube was inserted to give high oxygen concentration and artificial respiration when needed. The following day an encapsulated temporoparietal abscess was removed. Postoperatively the patient remained comatose and required continuous ventricular decompression. The tongue and pharyngeal muscles were completely relaxed so that during changing of the endotracheal tube there was apnea due to obstruction. His condition was too precarious to permit even the minimal trauma of tracheotomy until five days postoperatively. For about eight weeks profuse secretion necessitated frequent aspiration. Then the patient improved rapidly, with return of swallowing and cough reflexes. Decannulation of the tracheotomy was accomplished 10 weeks after operation, and the patient was discharged.

Comment.—This patient required assistance with respiration from the time of admission. An endotracheal tube was ideal, being easily inserted and used for

positive oxygen pressure or anesthesia. When artificial respiration was no longer necessary, tracheotomy was performed to prevent inflammatory reaction of the larynx as well as furnish a clear airway and simple means of aspiration.

CASE 11.—P. D., a white man aged 61, had a prefrontal lobotomy Sept. 5, 1949 to give symptomatic relief from recurrent carcinoma of the tongue which caused severe pain, inanition and bleeding from ulcerated areas. Postoperatively the patient was comatose, with a grayish pallor not relieved by oral airway, aspiration and oxygen. Respirations were very shallow and breath sounds distant throughout the chest. To facilitate aspiration and to by-pass the foul, crusted areas in the mouth, tracheotomy was performed. With the trachea open, very large amounts of mucoid blood-stained secretion were aspirated. At once the patient's color improved, and before leaving the operating table he was moving about spontaneously for the first time since lobotomy. Recovery was rapid thereafter, and the patient was discharged September 25 in a comfortable condition. The tracheotomy tube was kept in position as a safety measure for fear of hemorrhage from the mouth lesions.

Comment.—Since bronchoscopy was inadvisable because of the masses of malignant tissue in the mouth which bled with slight trauma, tracheotomy was done. When anoxia was relieved, cerebral depression rapidly disappeared.

The next two cases are similar and are presented together.

CASE 12.—M. B., a Negro boy aged 4, had a craniotomy Sept. 16, 1949 under endotracheal anesthesia, with partial removal of a frontal glioma. During the first 12 hours after operation obstructive laryngeal dyspnea developed. Direct laryngoscopy showed swelling, hyperemia and exudate over the larynx, with subglottic edema sufficient to embarrass the airway seriously. Insertion of a bronchoscope relieved the symptoms, and tracheotomy was performed. The patient recovered uneventfully, and decannulation of tracheotomy was carried out before discharge on November 24. The larynx was normal at this time.

CASE 13.—R. D., a white boy aged 6, had a posterior fossa exploration Dec. 18, 1948 under endotracheal anesthesia, disclosing a malignant cerebellar tumor. Ten hours postoperatively stridor and suprasternal retractions developed. Direct laryngoscopy revealed normal motility but marked subglottic swelling. Passage of a bronchoscope relieved dyspnea, and tracheotomy was performed. Convalescence was uneventful, and in three weeks the larynx was normal. Decannulation was accomplished, and the patient was discharged Feb. 5, 1949.

Comment.—In two children, who had long operational procedures under endotracheal anesthesia, laryngeal obstruction developed in the immediate postoperative period. In both, subglottic edema was pronounced and was attributed to use of the endotracheal tube. The patients' ages were 4 and 6 years respectively, and children, for anatomic reasons,[7] frequently show laryngeal obstruction when comparable insult in adults does not cause symptoms. Tracheotomy was essential, as the rapid deterioration pictured would soon have terminated fatally. Both larynxes soon returned to normal.

The next two cases are also presented together.

CASE 14.—E. R., a white woman aged 57, had a cerebral arteriogram Nov. 4, 1948 under thiopental (pentothal®) sodium anesthesia. After injection of the dye, there was some bleeding from the carotid puncture site. Despite local pressure, a mass as large as an orange appeared, and laryngeal obstruction suddenly developed. Attempts to pass an endotracheal tube were unsuccessful because the larynx could not be properly exposed, and tracheotomy was done through a stab wound incision. Although the patient regained consciousness shortly, an adequate oral airway did not appear until 10 days later, when the extravasated blood had been

7. Jackson, C., and Jackson, C. L.: The Larynx and Its Diseases, Philadelphia, W. B. Saunders Company, 1937, p. 113.

largely absorbed. At this time the larynx appeared normal, and the tracheotomy tube was removed. Later a frontal meningioma was successfully removed.

CASE 15.—V. W., a white woman aged 37, had a percutaneous cerebral arteriogram March 3, 1949 under thiopental sodium anesthesia. About five minutes after injection of the dye, the patient became cyanotic and stopped breathing. Strong respiratory efforts and suprasternal retractions indicated laryngeal obstruction, so an emergency tracheotomy was done. In a few minutes the vital signs were normal, and the patient made an uneventful recovery. The arteriogram showed an intracranial aneurysm, which was ligated April 14. The patient has remained well.

Comment.—Thiopental is an anesthetic agent which should be used with caution for head and neck surgery because of the danger of laryngospasm.[8]

COMMENT

During the past few years tracheotomy has become a valuable adjunct in management of patients who are unable to maintain the upper air and food passages free of obstructing secretions. No longer regarded solely as an emergency measure for laryngeal obstruction, tracheotomy may be a carefully planned procedure to prevent or treat anoxia. This change in thinking has been influenced by the many favorable reports on tracheotomy in tetanus and bulbar poliomyelitis,[9] diseases in which obstructing secretions are a serious hazard. These oral and nasal secretions, while easily swallowed or expectorated by a normal person, can seriously handicap the unconscious patient. Tracheotomy provides an effective means of clearing the tracheobronchial tree until recovery of normal functions.

This report does not discuss the management of the vast majority of neurosurgical patients, in whom the conservative measures of frequent turning, hyperventilation and tracheal aspiration are effective. Also, patients with laryngeal edema attributed to endotracheal anesthesia represent a small fraction of the number anesthetized by this method. However, cases do occur in which death from pulmonary complications seems inevitable postoperatively. Tracheotomy is presented as a method of combating anoxia in this type of patient. Five of the patients discussed here died in the hospital, but only one succumbed to pulmonary infection. This patient (case 5) expired from a combination of cardiac failure, senility and cerebral trauma in addition to hypostatic pneumonia.

Tracheotomy was employed when there seemed to be danger of sudden pulmonary obstruction, such as aspiration of vomitus or pharyngeal secretions. When pulmonary obstruction was already present, tenacious secretions could be removed. In addition, instillation of antibiotic solutions directly into the affected area was used to reenforce parenteral therapy. Obstruction in the larynx or pharynx was by-passed, and an oxygen catheter in the tracheotomy tube administered high oxygen concentrations when necessary. In these severely depressed patients, tracheotomy

8. Everson, S.: An Evaluation of Nitrous Oxide and Pentothal Sodium Anesthesia in the Practice of Oral Surgery, Oral Surg., Oral Med. & Oral. Path. **2**:24-32 (Jan.) 1949.

9. Godman, H. E., and Adriani, J.: Management of Patients with Tetanus: Some Clinical Experiences with Various Muscle-Relaxing Agents, J. A. M. A. **141**:754-756 (Nov. 12) 1949. Creech, O.; Woodhall, J. P., and Ochsner, A.: Necessity for Tracheotomy in the Treatment of Tetanus to Prevent Lethal Respiratory Complications, Surgery **27**:62-73 (Jan.) 1950. Galloway, T. C., and Seifert, M. H.: Tracheotomy in Poliomyelitis: Favorable Results in Its Treatment as a Problem in Respiratory Obstruction, J. A. M. A. **141**:1-8 (Sept. 3) 1949. Glaser, D. F.: Tracheotomy in Bulbar Poliomyelitis, J. Pediat. **27**:560-564 (Dec.) 1945.

greatly facilitated nursing care, and more time could be devoted to other aspects of the case.

Indications for tracheotomy in neurosurgical patients may differ from the classic dyspnea of laryngeal obstruction, and with marked cerebral depression the usual signs may be entirely absent. A high mortality results if the obstructive etiology of anoxia is not appreciated, and many factors must be considered in addition to laryngeal or pulmonary obstruction, such as (1) relaxed pharyngeal musculature, (2) loss of swallowing and gag reflexes, (3) thick crusts in pharynx or larynx secondary to the drying action of mouth breathing or continuous nasal administration of oxygen and (4) presence of feeding tubes over long periods acting as a foreign body irritant to the larynx with inflammation and even perichondritis.[10]

Patients in a diminished state of consciousness secondary to organic central nervous system disease may be unduly sensitive to even slight degrees of oxygen want.[11] Therefore it is particularly important to make an early estimate postoperatively of the patient's general pulmonary status and the cause of any oxygen deprivation. Changes in respiration may be distorted by the neurological disease, and even cyanosis is not always a dependable index of the degree of pulmonary obstruction. This is especially true in atelectasis, for cyanosis depends on an absolute amount of reduced hemoglobin, and in complete collapse there is very little blood passing through the nonaerating lung.[12] If there is any question of anoxia at clinical examination, examination of arterial blood oxygen saturation levels is the best method of accurate evaluation.

Whether or not tracheotomy should be performed must in each case be decided by close cooperation between the neurosurgeon and the bronchologist. If anoxia is present and does not respond to the usual methods of clinical treatment, tracheotomy should be considered as a proved means of avoiding further complications and adequately oxygenating the circulation. In our experience, patients who are severely enough depressed to lose the cough reflex over a considerable period frequently require tracheotomy, and even then pulmonary infection is difficult to prevent.

SUMMARY

Fifteen neurosurgical patients with complications requiring tracheotomy are described.

Immediate postoperative sequelae of intracranial surgery are (1) depressed cough and gag reflex, (2) obstruction by relaxed tongue and pharyngeal muscles, (3) tendency to aspirate solids and liquids and (4) rapid recurrence of respiratory obstruction in spite of frequent turning, tracheal aspiration and hyperventilation.

In the patients of this group, respiratory complications included atelectasis, aspiration pneumonia and laryngeal obstruction.

Some of the difficulties in evaluation of patients are mentioned.

The neurosurgical care of the patients reported was under the supervision of Dr. F. C. Grant and the bronchologic care under the supervision of Dr. J. P. Atkins.

10. Iglauer, S., and Molt, W. F.: Severe Injury to Larynx from Indwelling Duodenal Tube: Case Reports, Ann. Otol., Rhin. & Laryng. **48**:886-904 (Dec.) 1939.

11. Galloway, T. C.: Anoxia, Tr. Am. Laryng. A. **67**:41-56, 1945-1946.

12. Keeley, J. L., and Gibson, J. G.: Experimental Atelectasis in Dogs: Its Effect on Plasma Volume, Hemoglobin, Hematocrit, Blood Gases, Circulation Time and Pulmonary Blood Flow, Surgery **11**:527-543 (April) 1942.

BRONCHOSCOPY IN THE NEWBORN

The Otolaryngologist's Contribution to Obstetrics

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RESUSCITATION of the newborn infant has always been a major problem to obstetricians. Many devices have been used, including stimulation by immersion in hot and cold water, mouth to mouth breathing, the administration of carbon dioxide and oxygen, drug stimulation (methyl alpha-lobeline, caffeine, nikethamide [coramine], pentylenetetrazole [metrazol®]), suspension of the infant with the head down, the application of ether to, and blowing on, the body, smacking the feet and folding the infant, somewhat in the manner that an accordian is manipulated, slapping, artificial respiration, cleansing of the nose, mouth and larynx and the use of the catheter in an attempt to aspirate the trachea.

This last-named method, while it represents a practical approach to the problem, is inadequate. I realize that some experienced obstetricians have especial confidence in this method, but I have never been especially impressed by it. It is a blind approach. The catheter is more likely to enter the esophagus than the larynx and the trachea, and, if some fluid is aspirated, a false feeling of confidence is thereby engendered in the obstetrician. I believe, also, that the impact of the catheter on the unanesthetized larynx is apt to provoke laryngeal spasm and, thereby, increase the anoxemia of an infant already in jeopardy. I have not found it practical to introduce the catheter into the glottis by palpation. Finally, I have seen one patient who had hyperemia and edema of the larynx, which I thought was due to repeated jostling of the larynx by a catheter.

In regard to the use of mechanical resuscitation devices, employing positive and negative pressure, Goff,[1] in 1949, has especially deprecated their use, believing that they do more harm than the total of all other methods.

For these reasons, and for others to be mentioned below, such indirect methods of respiratory restitution should be replaced, whenever possible, by the direct method of bronchoscopic aspiration.

There are relatively few reports of bronchoscopic aspiration in the newborn. Woodward and Waddell,[2] in 1943, reported the use of bronchoscopy in the treatment of atelectasis in five newborn infants. In 1946, House and Owens [3] published

Read before the Ear, Nose and Throat Section of The International College of Surgeons Convention, Cleveland, Nov. 2, 1950.

1. Goff, W. F.: Respiratory Emergencies in the Newborn, West. J. Surg. **57**:49, 1949.

2. Woodward, F. D., and Waddell, W. W., Jr.: Bronchoscopy in the Newborn Infant, J. Pediat. **23**:79, 1943.

3. House, H. P., and Owens, P.: Atelectasis of the Newborn: Treatment by Bronchoscopic Drainage, J. Pediat. **28**:207, 1946.

their results in the treatment of 23 newborn infants with atelectasis by bronchoscopic drainage. Fifteen infants recovered. Most of the eight deaths were of infants with serious congenital defects or pathological processes of the central nervous system. Heatly and Emerson,[4] in 1948, analyzed 50 cases of bronchoscopy that they had performed in newborn infants. Thirty-five of these infants recovered. Cerebral hemorrhage, vascular anomalies, diaphragmatic hernia, massive atelectasis unassociated with bronchial obstruction, pneumonia and prematurity were the cause of death, as determined by autopsy, in the 15 cases of fatality.

The most recent publication is by C. C. Roe Jackson,[5] in 1950, who, after a detailed discussion of the subject, concluded that bronchoscopy can be done on all newborn babies and many premature babies with safety and that greater cooperation between obstetricians, pediatricians and otolaryngologists is necessary for the welfare of the newborn.

The purpose of bronchoscopy in the newborn is to resuscitate those infants whose inability to achieve adequate ventilation is due to laryngotracheobronchial obstruction. This condition may be due to a laryngeal web, a curled flaccid epiglottis, congenital stenosis of the larynx or paralysis of a vocal cord. Oftener, the presence of amniotic fluid, mucus, blood, meconium or vernix caseosa is the obstructive agent in these small passages.

Holinger, Johnson and Zoss [6] have emphasized that extensive pulmonary changes may be produced in the newborn, owing to the small caliber of the trachea and bronchi, by a quantity of foreign material that would be insignificant in a larger child.

It has been shown by Patterson and Farr [7] that there is a tidal flow of amniotic fluid throughout the bronchial tree and alveoli *in utero*. In this connection, Snyder and Rosenfeld [8] demonstrated the presence of india ink in the alveoli of animal fetuses, the transfer having been made from amniotic sac injection.

Heatly and Emerson [4] believe that in breech presentations, versions and cesarian sections, amniotic fluid, which is always present in the alveoli of the human fetus, is not so apt to be expressed because the compressive movements of the passage from the uterus through the birth canal are absent.

Bronchoscopy in the newborn serves to remove the fluid and any material aspirated during an otherwise normal birth. It also serves to detect such anomalies as tracheoesophageal fistula and agenesia of a bronchus. The cases to be selected are not those with failure of respiration, due only to central arrest from birth anesthesia or other medications, and intracranial trauma, but, rather, those with obstruction of the respiratory tract. I feel that it is a lifesaving mechanism in suitable cases, against which there is no reasonable argument. A direct lighted approach, with working accessibility, is superior to blind and indirect methods of resuscitation.

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4. Heatly, C. A., and Emerson, E. B., Jr.: Bronchoscopy of the Newborn: An Analysis of 50 Cases, Ann. Otol., Rhin. & Laryng. **57**:802, 1948.

5. Jackson, C. C. R.: Bronchoscopy in the Newborn, Ohio State M. J. **46**:869, 1950.

6. Holinger, P. H.; Johnson, K. C., and Zoss, A. R.: Tracheal and Bronchial Obstruction Due to Congenital Cardiovascular Anomalies, Ann. Otol., Rhin. & Laryng. **57**:808, 1948.

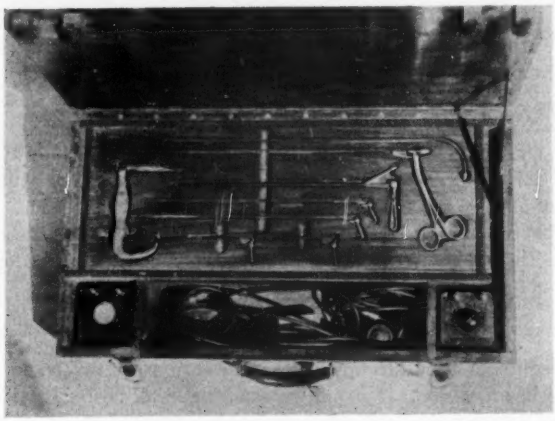
7. Patterson, J. C., and Farr, J. T.: Pulmonary Collapse as a Cause of Neonatal Deaths, Canad. M. A. J. **41**:31, 1939.

8. Snyder, F. F., and Rosenfeld, M. J.: Intra-Uterine Respiratory Movements of the Human Fetus, J. A. M. A. **108**:1946 (June 5) 1937.

When respiratory failure in the newborn is apparent, one must attempt to distinguish whether or not it is due to central or local effects. With prolonged posterior or face presentations, or other conditions requiring instrumentation, respiratory arrest may be due to damage of the respiratory centers from actual trauma, resulting in edema, hemorrhage or even tears. Prolonged anesthesia of the mother, and, also, frequent or heavy doses of some drugs, e. g., morphine, may depress the respiratory centers.

Anoxemia from foreign material in the upper respiratory tract or from compression of the neck by the umbilical cord is quickly relieved by bronchoscopic aspiration and the administration of oxygen, provided that there is not serious damage to the central nervous system from the anoxemia.

The insufflation of oxygen does not greatly relieve cyanosis of those infants with central arrest of respiration; however, it temporarily relieves those newborn



Equipment and container for bronchoscopy of the newborn, as maintained in the nursery.

infants with partial bronchial occlusion and atelectasis. By this, I do not mean to imply that oxygen therapy should not be administered continuously to those babies with central arrest, for I believe that oxygen, administered directly into the trachea through a bronchoscope, is a life-sustaining influence.

With the cyanotic and dyspneic infant, whose upper respiratory tract has been cleared of obstructive material, immediate partial improvement following the oronasal administration of oxygen suggests the presence of partial tracheobronchial obstruction. In such infants, the usual symptoms and signs are progressive dyspnea and cyanosis, aggravated by effort of any type. Other signs and symptoms, such as suprasternal retraction, with diaphragmatic tugging of the lower ribs, decreased thoracic expansion, and, sometimes, coarse, moist rales, are the signs known to most otolaryngologists. The history of aspiration of blood, amniotic fluid or other material certainly points to the possibility of partial tracheobronchial obstruction. If time and facilities permit, roentgenographic studies should be done. Sometimes, in those cases in which one sees newborn infants in oxygen apparatus

a number of hours after birth roentgenographic studies have been secured. In these infants, there has been cyanosis and dyspnea from birth, relieved by oxygen therapy and aggravated by its withdrawal.

The criteria of roentgenographic diagnosis of pulmonary atelectasis is so well known to all that it will not be discussed here.

Finally, if the diagnosis of aspiration and atelectasis in the newborn cannot be made with certainty, I can see no serious reason for not using bronchoscopy in any questionable case. The procedure is simple, quick and, if done with average skill, should be harmless. In the procedure, as pointed out above, anomalies and motor paralysis of the larynx will be detected. Since oxygen is always administered through the bronchoscope, the bronchoscopy is a supportive as well as a therapeutic procedure.

For the procedure, it is most practical to keep all the instruments immediately available on the obstetrical floor near the newborn nursery. For this purpose, my associate, Dr. J. C. Hays, and I have constructed a portable case, with slotted channels for each of the instruments and their duplicates. These are contained within a special compartment, with an "icebox" type of seal, in which there are formaldehyde tablets. The case also contains battery boxes, cords, space for bulbs, etc. The instruments can be withdrawn quickly and used. We customarily use the Holinger 3 mm. newborn infant bronchoscope, the Chevalier Jackson infant laryngoscope and a fine aspirator. This bronchoscope can be introduced into all larynxes of normal caliber without trauma, the tracheobronchial tree quickly explored and all the principal bronchi aspirated. The procedure should not traumatize the tissues. No especial postoperative care is necessary, except to watch for evidence of recurrence of the pulmonary condition or of glottic obstruction. I have not seen such obstruction develop.

SUMMARY

The procedure of bronchoscopy of the newborn is relatively new. It is an important salvage measure. It should be available in all hospitals with obstetrical services. Pediatricians, who are oftenest called to see the newborn with atelectasis, should be familiar with the clinical pattern and the relatively easy method of treatment. Obstetricians are counseled to use the scientific direct visual method of bronchoscopy for restitution of the lower airway. All otolaryngologists are urged to familiarize themselves with this technical procedure.

The cooperation of these three groups of specialists in this problem will lower the morbidity and the mortality rate of the newborn. The procedure is practical, sensible, safe and lifesaving.

JUVENILE NASOPHARYNGEAL ANGIOFIBROMA

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ADVANCES in therapy always constitute a good reason for presenting a familiar subject. Indeed, the chronology of juvenile nasopharyngeal hemangiofibroma reflects this steplike progress in the almost cyclical reappearance of reports in the otolaryngological literature as improvements in handling this sometimes alarming condition manifest themselves.

In reviewing the literature, I received the impression that a greater number of these cases were reported in the foreign journals, European and South American, than in those of this country. Part of this may be due to the depressant effect of the last war on medical literature in general, especially as regards the number of reports from this country compared with that of South America.

It has been the privilege of very few men to see several of these interesting cases, most reports being limited to one, two, three, four or six. In fact, when larger numbers of cases are reported often cases are included which may be open to dispute by reason of their occurring in women, in men beyond the usual age incidence, etc. The only large groups reported are those of Figi[1] and Hayes Martin and associates.[2] It is the latter, i.e., Martin and associates, who have attempted to lay down rather hard and fast rules for classifying this condition.

In their study they made the following observations:

1. All hemangiofibromas occurred in males. In five cases occurring in women on careful scrutiny the growths were found to be other types of tumor.

2. These tumors occur during the pubescent stage, usually at about 14. The word "juvenile" used in the descriptive title of this condition is incorrect, rather it should be called adolescent nasopharyngeal hemangiofibroma.

3. Regression of the tumor sets in when maturity is reached—generally at from 20 to 25 years of age.

Thus, a nasopharyngeal tumor which does not fulfil these requirements must be looked on with suspicion. It is true that the microscopic picture should assist greatly in the differential diagnosis, but owing to the fact that the taking of a biopsy is frequently attended by profuse hemorrhage it is often felt advisable to irradiate the tumor initially to lessen its vascularity. Figi[1] in his article admits

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Presented before the Chicago Laryngological and Otological Society, Chicago, April, 1950.

From the Department of Otolaryngology, Stritch School of Medicine of Loyola University and Mercy Hospital.

1. Figi, F. A.: Fibromas of the Nasopharynx, J. A. M. A. **115**:665-671 (Aug. 31) 1940; The Treatment of Hemangiomas of the Head and Neck, Plast. & Reconstruct. Surg. **3**:1-10, 1948.

2. Martin, H.; Ehrlich, H. E., and Ables, J. C.: Juvenile Nasopharyngeal Angiofibroma, Ann. Surg. **127**:513-535, 1948.

that in some cases he is willing to base a diagnosis on the clinical appearance of the tumor, its course and response to irradiation therapy rather than to take a biopsy of the growth.

Seen under the microscope at different periods in their growth, these tumors, which are essentially vascular fibromas, show varying amounts of endothelial blood lakes and fibrous stroma. Fibromas located anywhere in the body tend to become less vascular and more fibrous as age increases. Thus, the "early" and "late" microscopic observations are at variance. Necrosis following infection, ulceration and thrombosis alters the picture and sometimes renders diagnosis from the pathologist's standpoint difficult. Often, especially in the past, these tumors have been mistakenly cataloged under myxoma, sarcoma, giant cell tumor, etc.

CLINICAL OBSERVATIONS

Defining adolescent nasopharyngeal hemangiofibroma as an entity, it may be said to be an unencapsulated, usually hard, rounded or lobulated, noinfiltrating, extremely vascular benign tumor found in the nasopharyngeal vault of youths, and it has a tendency to regress as maturity is reached.

Symptoms.—The symptoms depend on the size of the tumor, rate and direction of growth and efforts to halt its progress. It is conceivable that some of these neoplasms go unrecognized because of late start, slow expansion or early regression. One such case is reported in the literature. It was found during a routine nasopharyngeal examination. Unilateral nasal obstruction, which occurs when the nasopharyngeal portion of the tumor has attained a size of about 3 cm., is usually the earliest sign and may be present for about a year before help is sought. Occurring as it does in boys whose attention is distracted from such minor complaints, nasal obstruction is rarely heard of—rather the presenting complaint is usually hemorrhage, and the previous history of obstruction is elicited on questioning. Ulceration and necrosis of the tumor usually follow repeated tamponade in attempts to arrest the hemorrhage. Voice change (rhinolalia clausa) is noticed when the tumor is large enough to fill the entire nasopharynx, and various grades of otitis media with impairment of hearing on the affected side appear as the growth expands. Involvement of sinuses due to impaired ventilation and later actual invasion by the tumor varies from showing slight clouding on the roentgenogram to frank purulency. Neuralgia, dysphagia, proptosis and facial deformity are dependent on the extent of the growth. "Frog face," a grotesque facial appearance occurring in the course of many nasopharyngeal tumors, is mentioned frequently in the earlier literature on this subject. Anemia and toxicity are frequent accompaniments, and intracranial involvement is a possibility.

Diagnosis.—The diagnosis depends, of course, on a history of any of the above symptoms in a youth and the finding of a tumor by posterior rhinoscopy in the nasopharynx. It is usually smooth and covered with an apparently normal mucosa which may or may not show some enlarged superficial vessels. Fullest inspection is achieved by topical anesthesia and some means of palate retraction. If the mass has extended forward into the naris, it may be apparent on anterior rhinoscopy. Use of the nasopharyngoscope may lend some added details.

Palpation is very useful in determining the extent of the attachment of the mass. General anesthesia is more than justified if it is necessary to carry out these procedures. Lateral roentgenogram views of the neck—flat plate and stereoroentgenogram—are helpful in further delineating the mass. A biopsy whenever possible should be taken. Precautions for control of hemorrhage, including facilities for transfusion, should be available.

Pathology.—The broad base of origin from which this tumor springs and which is described so well by Brunner [3] is best referred to as the basal fascia or pharyngeal aponeurosis of the superior constrictor muscle, which blends with the buccopharyngeal fascia and the periosteum of the basilar portion of the occiput, upper two cervical vertebrae and the sphenoid bone. This vascular region and the presence of immature connective tissue in the midline, as claimed by some, no doubt contribute to the rapid growth of the tumor, as has been pointed out so often. Some tumors reach a size of 10 to 12 cm. in diameter and may contain fragments of bone on their surfaces from eroded nearby structures. There are no metastases. Malignant transformation, though reported, is questionable. Recurrences following incomplete removal are the rule. The return is rapid and the secondary growth of large size (contrary to Ewing [4]).

Microscopic Appearance.—Stroma with round or stellate fibroblasts and large endothelial lakes resembling the cavernous variety of hemangioma are apparent. This is the early, or soft, type. There is also a later type which shows sclerosing changes, lessened vascularity, hyalinization, a pseudocapsule and firm consistency. There is no elastic tissue (Gross and Wolbach [5]), and the vessels have no muscle in their walls. Areas in the same tumor vary. Immature fibroblasts frequently are so dense as to suggest sarcoma. Foci of lymphocytes and plasma cells may obscure the true picture. Fragmentation of endothelial walls by hyalinization may give rise to large numbers of giant cells, and fatty degeneration may accompany necrosis. Brunner [3] from his study of serial sections points out that the angiomatous vessels of the tumor communicate with veins of the surrounding normal tissue but not with the arteries. This may help to account for the frequent referral in reports of cases to the fact that ligation of one or both carotids did not seem to lessen the tendency of the tumor to bleed during manipulation (Munson [6]).

Treatment.—As indicated in the opening paragraph, treatment has been undergoing constant revision with resultant improvement. Surgical extirpation, long the only method available, is far from ideal. Because of the real or fancied multicentric origin, complete removal in such a disadvantageous position is next to impossible. The avenues of approach (Brown [7]) by Moure's lateral rhinotomy and Denker's

3. Brunner, H.: Nasopharyngeal Fibroma, Ann. Otol., Rhin. & Laryng. **51**:29-65, 1942.

4. Ewing, J.: Neoplastic Diseases: A Treatise on Tumors, ed. 4, Philadelphia, W. B. Saunders Company, 1940.

5. Gross, R. E., and Wolbach, S. B.: Sclerosing Hemangiomas: Their Relationship to Dermatofibroma, Histiocytoma, Xanthoma and to Certain Pigmented Lesions of the Skin, Am. J. Path. **19**:533, 1943.

6. Munson, F. T.: Angiofibroma of the Left Maxillary Sinus, Ann. Otol., Rhin. & Laryng. **50**:561-569, 1941.

7. Brown, J. M.: The Surgical Treatment of Nasopharyngeal Fibroma, Ann. Otol., Rhin. & Laryng. **56**:294-297, 1947.

operation, or the transpalatal route, do not give the desired exposure and deforming scars are left. Recurrences are common, and access to these is difficult by the same path.

Coagulation by any method of cautery or combined with surgical removal, as with a snare activated with short-wave diathermy, is also incomplete and attended with primary hemorrhage which is difficult to control. Sclerosing solutions given by injection add the element of infection and are of indeterminate action. Radiation may interfere with the growth centers in the adjacent bones and if applied in sufficient amounts causes atrophy of the mucosa of the nose and throat producing the familiar symptoms of dryness and crusting.

Hormone therapy is an intriguing newer agent for combating this tumor. Alexander Lipschütz and associates [8] from Chile have shown experimentally in guinea pigs that most estrogens, or female hormones, will produce multiple fibromas if injected over a sufficient period and in such a manner as to maintain a fairly constant level of the estrogen in the body. These fibromas are not only uterine but also extrauterine or abdominal, i. e., along the intestinal tract, on the liver, etc. These experimentally produced fibromas regress if the estrogen is discontinued and injection of the male hormone, or androgen, is begun.

While these fibromas differ from those occurring in the uteri of women, they are like them in that they regress when production of estrogen is suspended or diminished, as in the menopause or after castration or ovarian irradiation. There is a good deal of evidence to support the theory that uterine fibromas in women are an endocrine problem involving the adrenal gland.

The fibromatogenic or antifibromatogenic action of the steroid hormones varies according to the species of animal. Estradiol does not produce fibroids in the rat yet does so easily, as indicated above, in the guinea pig.

Martin and associates [2] were unable to induce regression by sex hormone therapy alone, but they felt that it enhanced radiation to a point at which smaller doses of the latter were required to control the growth. Thus, it appears that the combination of radiation and androgen therapy is the method of choice for smaller lesions. Larger lesions, especially those producing deformity, require a combination of electrosurgery and hormone therapy with implantation radiation or cautery or both for small recurrences.

Incidence.—This is the first case seen by me in my own private practice. At Mercy Hospital the last patient admitted with this diagnosis was 10 years ago. The records at Cook County Hospital for the past 10 years revealed 10 cases, but these included five women and two of the men were well beyond their thirtieth year. A survey of the pathological slides in these cases was not attempted.

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8. Lipschütz, A.; Vargas, L., Jr., and Nunez, C.: Comparative Antitumoral Action of Desoxycorticosterone and Testosterone Propionate, Proc. Soc. Exper. Biol. & Med. **48**:271-274, 1941. Lipschütz, A.; Bruzzone, A., and Fuenzalida, F.: Structural Characteristics of Antifibromatogenic Steroids: Experiments with Acetoxy-Pregnenolone, ibid. **54**:303-304, 1943. Iglesias, R., and Lipschütz, A.: Structural Particularities of Antifibromatogenic Steroids: Experiments with Δ^4-Androstene-3, 17-Dione and Cholestenone, ibid. **55**:41-43, 1944. Lipschütz, A.: Experimental Fibroids and Antifibromatogenic Action of Steroid Hormones, J. A. M. A. **120**:171-175 (Sept. 19) 1942.

Shaheen [9] reported 58 cases in 1930, but most writers following him feel that he included conditions other than hemangiofibroma. Figi [1] reported 63 cases over a period of 30 years seen at the Mayo Foundation, or about two a year (five were in women).

Martin and associates [2] enumerated 29 cases in a period of 20 years. At the same time about 2,000 cases a year of neoplasms about the head and neck are seen at Martin's institution, the Memorial Hospital, New York. Thus, in incidence as well as in behavior this is an unusual tumor.

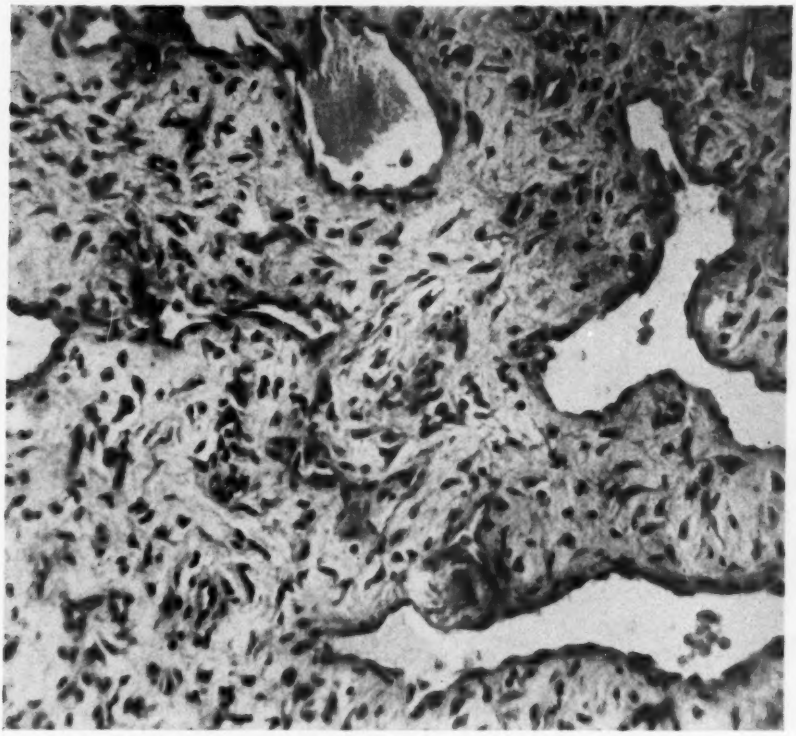

Fig. 1.—Section from untreated tumor taken Feb. 28, 1949 (× 320).

REPORT OF A CASE

On Feb. 26, 1949, I was asked to see a patient at Mercy Hospital with an active epistaxis, a white youth 16 years of age, somewhat "washed out" in appearance from both loss of blood and lack of sleep. His story was that the epistaxis, which was from the left naris only, had begun spontaneously three days previously, and that the family physician had used an anterior pack of petrolatum gauze with temporary success. Resumption of the bleeding sent him to a second physician and finally a third. None of these men apparently suspected the nature of the case, as conversation with them later on bore out. Inspection by anterior rhinoscopy revealed a mass lying alongside the inferior turbinate bone and slightly above. It was about

9. Shaheen, H. B.: Nasopharyngeal Fibroma, J. Laryng. & Otol. **45**:259-264, 1930.

a half inch from the vestibule. An ulceration about 3 to 4 mm. in diameter was the site of the bleeding. A pledget of oxycel® cotton saturated with prothrombin solution brought most of the bleeding under control in a relatively short time.

Inspection of the pharynx to look for blood going down this avenue revealed the soft palate a little low and bulging especially on the left side. Posterior rhinoscopy showed a rounded mass covered with normal-appearing mucosa filling almost the entire epipharynx. Further exposure with a Haslinger palate retractor after topical anesthesia revealed the mass as originating from the left vault and upper posterior septal edge. It appeared to be about 5 cm. in diameter. There was a cleft separating it from the lateral wall, the soft palate and lower portion of the septum. The right posterior choana was only partially occluded by a

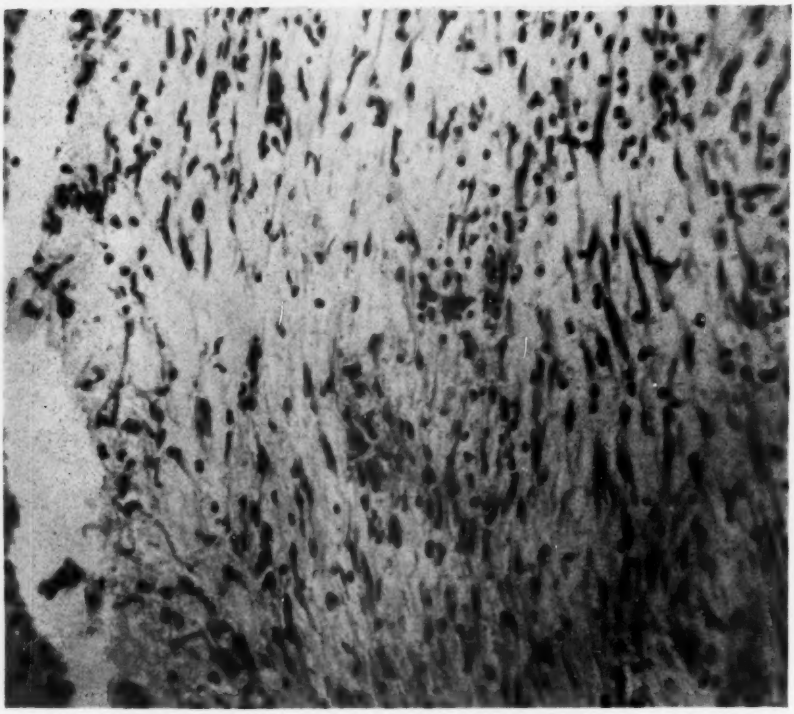

Fig. 2.—Section through mass near end of irradiation therapy and while the patient was receiving testosterone therapy, taken March 21, 1949 (× 330).

lobulation of the mass which extended around the septal edge. The youth was put to bed in the hospital and the administration of coagulants, calcium gluconate and vitamin K was begun.

During the next two days bleeding was minimal and infrequent, allowing an opportunity for other investigations. Roentgenograms revealed clouding of all the sinuses on the left side, and blood study revealed but a moderate degree of anemia. Physical examination revealed an apparently normally developed youth. The testes were descended, and there was no infantilism of the genitalia.

Under general anesthesia induced by intravenous administration of thiopental (pentothal®) sodium and supplemented with gas intratracheally, examination by palpation was undertaken by the oral and posterior pharyngeal route and through the anterior naris, with combination of the two from time to time. Contrary to the usual case description in the literature of a firm,

almost cartilaginous, tumor, this was soft and easily reducible; so much so that any attempt at removal by grattage would have been impossible. The attachment was extremely broad and mainly to the roof of the nasopharynx. It apparently was pear shaped, with the base in the nasopharynx and the stem portion in the left nostril. A biopsy was taken, and the bleeding that ensued was brisk. It again was fairly well controlled by an anterior pack. The biopsy report confirmed our clinical suspicions. The pathologist defined the tumor as a fibroma with multiple simple endothelium-lined blood spaces covered by intact mucosa. Sections are shown with the dates at which they were taken (figs. 1, 2 and 3).

Dr. P. R. Nelson of the department of radiology was consulted, and he advised external roentgen irradiation through two ports, over each maxilla. The factors were 220 v. (primary),

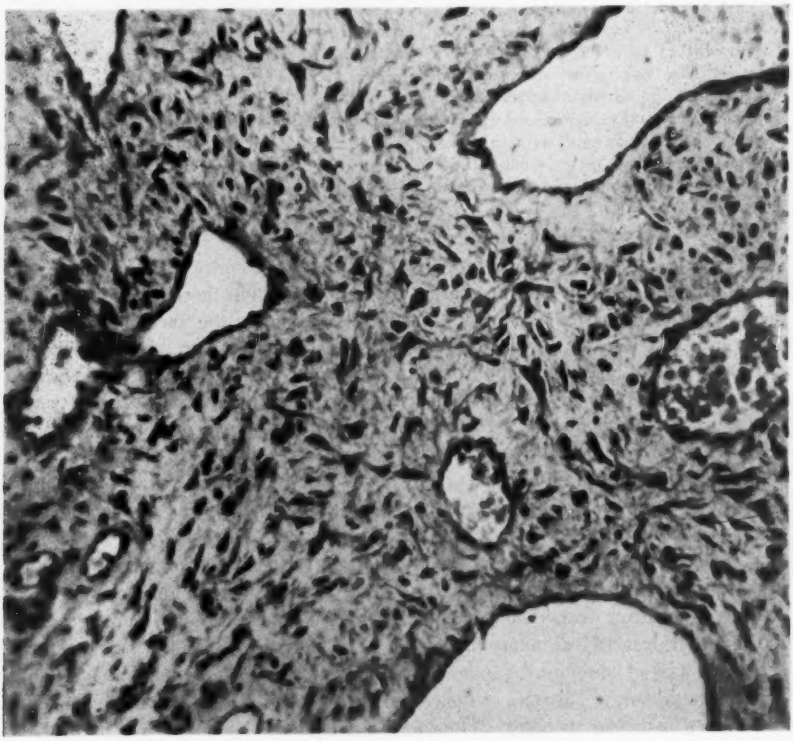

Fig. 3.—Section from mass one year later (× 330). Very little bleeding attending the taking of this biopsy. The patient was still receiving testosterone therapy. No further irradiation therapy has been used.

200 kv., 25 ma., 1.0 mm. Cu and 1.0 mm. Al filtration, field size 10 × 15 cm., focal skin distance 50 cm., 250 r at surface of field, time in minutes per treatment 6.5., 8 treatments to right and 8 treatments to left nasopharynx from March 7, 1949 to March 29, 1949 inclusive.

Coincidentally with the radiation and other therapy, the daily administration of androgen in the form of testosterone propionate was begun. Ten milligram sublingual tablets (linguets®) were allowed to dissolve under the tongue or in the buccomandibular gutter so that the hormone would be absorbed from the mucosa and not undergo digestion, as it probably would by oral administration. A subcutaneous implant of the hormone to insure continuous absorption, which is undoubtedly a more ideal method of administration, was considered, but response was so favorable it was not made use of.

Biopsies were taken at two week intervals, and after about five weeks of hospitalization the youth was sent home and told to return weekly for outpatient observation. Bleeding had ceased completely by this time, and the original ulceration and biopsy sites were covered by mucosa. Two months later there was a recurrence of bleeding which continued intermittently for one week. It was scanty and followed a one mile hike under a hot sun. During the fourth and fifth months the patient noticed the gradual return of his airway on the left side. He began developing a beard on his upper lip, and he showed an increase in height and weight. The mass gradually withdrew at this same time and was no longer apparent on anterior rhinoscopy, and the portion in the posterior nasopharynx shrank to about 3 cm. in diameter, permitting a view of a cleft between the mass and most of the posterior septal edge, the soft palate and the lateral wall of the nose. A further shrinkage to about 1.5 to 2 cm. in diameter occurred. During the next two months the intake of testosterone was reduced gradually to two sublingual tablets daily. During the ninth and tenth months an increase in size to about 3 cm. was suspected. The number of tablets was doubled for a month, and when reduction in size occurred once again the dose was further increased and a biopsy taken. Very little bleeding followed this procedure. A simple cotton tampon left in place for 15 or 20 minutes completely staunched the bleeding. The section showed vessels of much smaller caliber than those in the original. At present the mass is as small as it has ever been, i. e., about 1.5 to 2 cm. The youth does heavy work and has had two or three upper respiratory tract infections with no recurrence of bleeding. His airway is unimpeded, and his psychological outlook, as well as that of his parents, is much improved.

COMMENT

It may be difficult to make this tumor stand out as an entity, since fibrous tissue occurs all over the body and neoplasms having their origin therein are affected by the locale. Thus, fibromas are multinamed depending on the variation of fibrous tissue involved, i. e., bone (osteoma), cartilage (chondroma), etc., or the admixture of other elements, i. e., lymphofibroma, angiofibroma, etc. However, none seem to have the factor of such a specifically timed regression. The only other example is the occasional spontaneous regression of uterine fibromas in women after the menopause (Som and Neffson[10]). The use of estrogens locally in the nose to combat what may be alluded to as the reverse of this, namely atrophic rhinitis, was brought out some 10 years ago by Soskin and Bernheimer.[11] The beneficial effect was thought to be induced by altering the amount of acetylcholine in the nasal mucous membrane. Work on laboratory animals relating to the cholinergic action of the estrogens has been done by Reynolds and Foster,[12] who pointed out the effect of these stimulating female hormones on mucous membrane. They, among others, showed that loss of secondary male sex characteristics must be looked for on prolonged use of amniotin® (an estrogenic preparation) or alpha-estradiol (progynon®) nasal spray. So, too, in the continued use of the androgens there is danger of inducing testicular atrophy. Thus, it is a therapeutic addition which will bear careful watching and is full of possibilities. Conservatism must be the watchword, because the consensus seems to be that mere attainment of maturity by these youths

10. Som, M. L., and Neffson, A. H.: Fibromas of the Nasopharynx: Juvenile and Cellular Types, Ann. Otol., Rhin. & Laryng. **49**:211-218, 1940.

11. Soskin, S., and Bernheimer, L. B.: Mechanism of Estrogen Effect on Nasal Mucosa in Atrophic Rhinitis: Successful Treatment with Prostigmin, Proc. Soc. Exper. Biol. & Med. **42**:223-224, 1939.

12. Reynolds, S. R. M.: Acetylcholine Content of Uteri Before and After Administration of Oestrin to Ovariectomized Rabbits, J. Physiol. **95**:258-268, 1939. Reynolds, S. R. M., and Foster, F. I.: Species Differences in the Cholinergic Action of Estrogen, Am. J. Physiol. **131**:200-202, 1940.

will result in cure. Too, if it can be shown that the use of androgens hastens the regression of this tumor in the youth, it is reasonable to hope that more efficient steroids will be developed in the future which may affect fibromas in general.

SUMMARY

Juvenile nasopharyngeal angiofibroma is a relatively rare tumor restricted apparently to adolescent males. It regresses as maturity (20 to 25 years) is reached. Epistaxis is the chief symptom and usually is serious. Ligation of the carotid is of questionable benefit. Surgical extirpation is almost impossible. Recurrences are common, and the secondary growth is rapid and large. Radiation and hormone (androgen) therapy offer the best possibilities of control. Large tumors producing deformity of skull (frog face) are resected by electrocoagulation and remnants or recurrences are treated with interstitial irradiation and androgen. The most conservative therapy is always employed because of the usual spontaneous regression of the tumor at maturity.

NONCHROMAFFIN PARAGANGLIOMA OF THE MIDDLE EAR (GLOMUS JUGULARE TUMOR)

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AND
R. N. GRANT, M.D.
NEW YORK

THE PURPOSE of this paper is to report two new cases of nonchromaffin paraganglioma of the middle ear and to present a review of the medical literature and a résumé of the newer concepts of the pathogenesis of this tumor.

REVIEW OF THE LITERATURE

In 1941, Guild [1] described a paraganglionic structure in the adventitia of the dome of the jugular bulb and along the ramus tympanicus of the glossopharyngeal nerve, resembling morphologically the carotid body, which he called the glomus jugulare. Watzka [2] recognized two types of paraganglions: (1) chromaffin, epinephrine-producing paraganglionic tissue and (2) nonchromaffin, nonepinephrine-producing paraganglionic tissue, the latter type associated with fibers of cranial nerves. In this second group he lists the carotid body, aortic bodies and "cells and cell groups found in the trunks of the glossopharyngeal and vagus nerves." The so-called glomus jugulare of Guild is one of the more recently described nonchromaffin paraganglions, and tumors derived from it are classified as "nonchromaffin paragangliomas." The function of these paraganglions is probably that of chemoreceptors able to detect changes in the p_H or the carbon dioxide concentration in the blood that circulates through their rich vascular networks.

Rosenwasser [3] in 1945 described a middle ear tumor with the histological characteristics of a paraganglioma of the carotid body type. He postulated that it had originated in the glomus jugulare and extended through the floor of the middle ear into the tympanic cavity. Since then, about 28 other similar middle ear tumors have been described in the literature. A few of these tumors, originally described as hemangiomas, hemangioendotheliomas or granular cell myoblastomas, have been reclassified as nonchromaffin paragangliomas of the middle ear. Five reported cases have terminated fatally with intracranial extension. In many of the cases with survival, as well as those with fatal termination, there was involvement

From the Head and Neck Department, Memorial Hospital, Service of Dr. Hayes Martin.

1. Guild, S. R.: A Hitherto Unrecognized Structure, the Glomus Jugularis in Man, Anat. Rec. (supp. 2) **79**:28, 1941.

2. Watzka, M., cited by Lattes, R., and Walner, J.: Nonchromaffin Paraganglioma of the Middle Ear, (Carotid-Body-Like Tumor; Glomus-Jugulare Tumor) Cancer **2**:447-468, 1949.

3. Rosenwasser, H.: Carotid Body Tumor of the Middle Ear and Mastoid, Arch. Otolaryng. **41**:64-67 (Jan.) 1945.

by extension of the seventh, eighth, ninth, tenth and twelfth cranial nerves, either singly or of several of these nerves. Lattes [4] has recently reported a case in which tumor occurred in several members of the nonchromaffin system at one time. Thus one may see nonchromaffin paraganglioma of the glomus jugulare and of the carotid body in the same patient, as in the case reported by Kipkie [5] and in one case of those reported by Lattes and Walner.[6] In two of the published cases metastases developed. In one of the cases reported by Winship [7] there was metastatic involvement of the ipsilateral (left) upper jugular node histologically proved. In one case of Lattes and Walner [6] with fatality, a large metastasis to the liver was found at autopsy.

REPORT OF CASES

CASE 1.—M. S., a 63 year old white woman, with the complaint of watery discharge from the left ear of six months' duration, was referred to the head and neck clinic of the Memorial Hospital because of the presence of a tumor in the left external auditory meatus. There had been gradually increasing deafness and constant buzzing in the ear since the onset but no pain. Two weeks before admission, snare removal of a mass in the left external auditory canal had been attempted elsewhere, but it was terminated because of profuse hemorrhage. Some tissue was obtained for examination, and on review of this material in the pathology laboratory of the hospital a diagnosis of glomus jugulare tumor was made.

Examination on admission to the clinic revealed grayish pink granular tissue that completely filled the external auditory canal and that was bathed in serous exudate. There was no evidence of cranial nerve abnormality or cervical metastases. An incidental finding was a 5 cm. cystic, nontoxic thyroid adenoma in the right lobe, which collapsed on aspiration of 30 cc. of straw-colored fluid. Roentgenographic examination of the mastoid processes disclosed only sclerosis of the left mastoid but no evidence of bone destruction.

She was admitted to the hospital, and on Oct. 10, 1950, a tympanomastoidectomy on the left was performed through an endaural approach. During the removal of the bony posterior canal wall there was moderate bleeding, but as the facial ridge was cleared, hemorrhage became brisk. The tumor filled the entire tympanic cavity and was removed intact from its bed (figs. 1 and 2). This procedure was followed immediately by profuse venous hemorrhage arising from the floor of the middle ear, presumably from the jugular bulb. The defect was packed with oxidized cellulose U. S. P. (oxycel® gauze) and narrow gauze packing. Nasopharyngeal bleeding from the eustachian tube was controlled by a postnasal pack, which was left in place for eight hours. A pressure dressing was applied. During the procedure the patient received 500 cc. of whole blood. Postoperatively, she received penicillin and aureomycin, and the packing was completely removed by the fifth postoperative day. She was discharged on the fifth postoperative day, and her course has been uneventful.

CASE 2.—R. L., an 80 year old white woman, with the complaint of fulness in the right ear of six months' duration, consulted her family physician because of slight bleeding from the ear. Biopsies were made elsewhere, and the diagnosis was "hemangioendothelioma." On examination at the head and neck clinic, gray, granular tumor tissue was found occluding the right external auditory meatus. There was no evidence of cranial nerve involvement or cervical metastases. Roentgenograms of the mastoids showed no gross abnormality. Biopsy at the Memorial Hospital

4. Lattes, R.: Nonchromaffin Paraganglioma of Ganglion Nodosum, Carotid Body, and Aortic-Arch Bodies, Cancer **3**:667-694, 1950.

5. Kipkie, C. F.: Simultaneous Chromaffin Tumors of the Carotid Body and Glomus Jugularis, Arch. Path. **44**:113-118 (Aug.) 1947.

6. Lattes, R., and Walner, J.: Nonchromaffin Paraganglioma of the Middle Ear (Carotid-Body-Like Tumor; Glomus Jugulare Tumor), Cancer **2**:447-468, 1949.

7. Winship, T.; Klopp, C. T., and Jenkins, W. H.: Glomus-Jugularis Tumors, Cancer **1**:441-448, 1948.

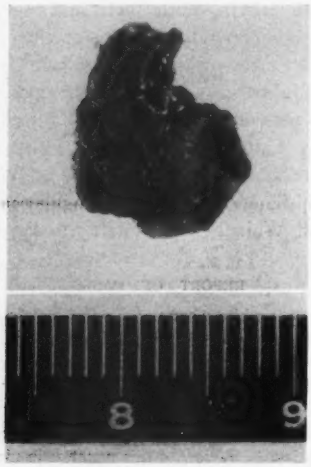

Fig. 1.—Section of tumor removed in case 1.

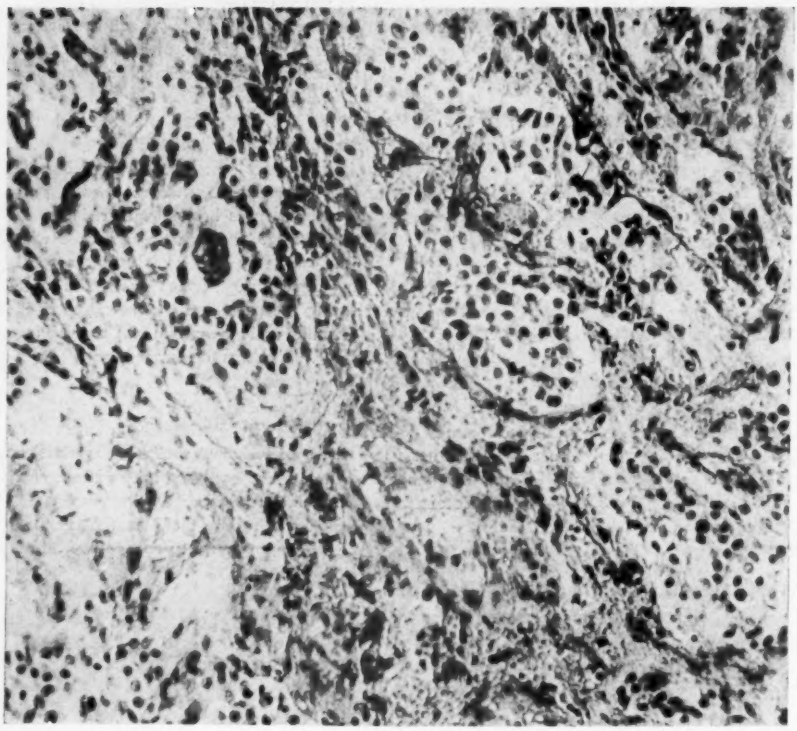

Fig. 2.—Specimen of tumor (case 1).

was followed by severe hemorrhage, which was controlled by gauze packing. The pathological diagnosis was glomus jugulare tumor.

Surgical extirpation was proposed, but the patient persistently refused operation. Thirty months after the original diagnosis was made, because of slight increase in the size of the aural tumor, a trial of low voltage roentgen ray therapy was given: 100 kv., 15 cm. target skin distance, 1 mm. Al filtration, 2 cm. portal and four doses of 300 r for a total of 1,200 r. This treatment had no visible effect on the tumor. Part of the tumor was removed by snare cautery, and the bleeding was controlled by gauze packing. The patient has been observed for 40 months, and no signs of tumor extension have been noted.

COMMENT

The ages in the reported cases ranged from 23 to 81 years, with a median age of 48. There has been a preponderance of female patients with this condition; of the 30 patients, 24 were women. The occurrence of 80 per cent of these tumors in women raises the question whether there might be some hormonal relationship, and a corollary suggestion arises that perhaps the use of androgenic hormones may modify the tumor, either preoperatively to decrease vascularity or in cases in which surgical excision is refused.

Because of the location of this type of nonchromaffin paraganglioma in the temporal bone, extension into surrounding structures is of serious consequence. Accordingly, early removal of the tumor is mandatory. Because of the rich vascularity of the tumor and its origin from the wall of the jugular bulb, considerable hemorrhage is produced on manipulation at the time of operation. Adequate exposure of the tumor through the mastoid is afforded by the endaural approach, which permits complete removal under direct visualization and access for the control of hemorrhage. The first patient described in this report was operated on through an endaural approach. The tumor completely filled the tympanic cavity and was removed en masse with ease (fig. 1). Bleeding was controlled by firm oxycel® gauze and gauze packing in the operative defect.

Because the pathogenesis of these tumors was not clarified until recently, only five cases reported in the literature have been followed five years or more. Of the patients with long term survival, one reported on by Kipkie [5] was observed for 10 years after radical mastoidectomy, only to succumb to a second operation for the removal of a carotid body tumor. At autopsy, it was found that the petrous pyramid was eroded by tumor tissue.

Winship [7] reported a case in which radical mastoidectomy was performed, and 12 years after the original diagnosis a proved cervical metastasis was found. Thirteen patients were observed for an average of 5½ years after radical mastoidectomy without evidence of tumor extension. Five other patients treated with local excision were observed for an average of 3½ years without signs of extension.

The nine patients reported who were treated with roentgen ray therapy demonstrated no appreciable radiosensitivity of these tumors regardless of the factors or dosage.

The second patient reported on in this paper showed no response to a trial of low voltage roentgen ray therapy. While the course of this patient has been benign up to the present, the experience derived from the previously reported cases indicates that extension into adjoining intracranial structures or the development of

metastases is a dangerous potentiality. Accordingly, early surgical removal appears to offer the best prognosis in the treatment of nonchromaffin paraganglioma of the middle ear and can be accomplished adequately only by means of a radical tympano mastoidectomy.

SUMMARY

Two cases of nonchromaffin paraganglioma of the middle ear are reported. In one, surgical removal of the tumor was made through an endaural tympanomastoidectomy.

The relationship of the "glomus jugulare tumor" to other nonchromaffin paragangliomas is reviewed.

The early surgical removal of these tumors is recommended because of their locally destructive nature and their metastasizing potentiality.

An endocrine relationship is suggested in the natural history of these tumors.

TOXICITY OF CERTAIN TOPICAL ANESTHETICS USED IN OTOLARYNGOLOGY

An Experimental Study

HENRY J. RUBIN, M.D.
LOS ANGELES

THE APPREHENSION with which otolaryngologists view the matter of topical anesthesia is well known. Differences in opinion and technic are so great as to suggest that the only single point of agreement is on the general hazards of anesthesia. Confusion is attributable partly to a lack of pertinent experimental work on animals. Although there are admittedly sharp limitations to the applicability of animal studies to man, virtually all that is known about the physiological action of these drugs is based on laboratory studies, and doses officially approved as safe for man were calculated directly from animal responses. However, most laboratory investigations on topical anesthetics have been pursued by injection methods, and the literature on toxicity of cocaine and tetracaine hydrochloride (pontocaine hydrochloride®) with topical application is sketchy.

Runge and Schmidt,[1] in one of the early articles on tetracaine, stated that topical application was similar to subcutaneous injection and that if the solution entered the tracheobronchial tree, absorption approached that of intramuscular injection. Experimental work from which these conclusions were drawn was not described.

Eggleston and Hatcher [2] introduced cocaine-soaked cotton pledgets into the noses of cats and found that convulsions were produced with 53 mg. per kilogram. However, they felt that some of the solution was swallowed. How much might have remained in the cotton without being absorbed is not indicated.

These reports on topical absorption are in striking contrast to the vast literature which has accumulated on toxicity investigations by other methods. The otolaryngologist has been compelled, for the most part, then, to draw conclusions as to the toxicity of cocaine and tetracaine from experiments in which the agents have been administered differently from the manner in which he uses them.

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Thesis approved by the faculty of the Graduate School of Medicine of the University of Pennsylvania in partial fulfilment of the requirements for the degree of Master of Medical Science [M.Sc. (Med.)].

1. Runge, H. G., and Schmidt, H.: Pantocain, ein vollwertiger Kokainersatz, Arch. f. Ohren-Nasen- u. Kehlkopfh. **128**:232, 1931.

2. Eggleston, C., and Hatcher, R.: Pharmacology of Local Anesthetics, J. Pharm. & Exper. Therap. **13**:433, 1919.

This study is concerned with the toxicity of cocaine and tetracaine when applied in a variety of circumstances to the mucous membranes of rabbits and guinea pigs. Solutions tested were cocaine, 10 and 50, and tetracaine, 0.5 and 2 per cent.

EXPERIMENTAL PROCEDURE

Rabbits weighing between 2 and 4 Kg. and guinea pigs weighing from 0.35 to 0.75 Kg. were used in these investigations. They were fed a stock diet. Animals given sublethal doses were used repeatedly after appropriate rest periods. Eventually, all were killed in determining minimum lethal doses.

Figure 1 demonstrates the manner in which rabbits are restrained for instillation of solutions into the nose. With the nasal passages lower than the oropharynx, no anesthetic can enter the throat, and the solution is introduced through a blunted

Fig. 1.—Position of rabbit for intranasal instillation of anesthetic solution.

needle just inside the anterior nares. The head may be moved from side to side to encourage diffusion. There is little or no resistance by the rabbit if the solution is introduced slowly, and no effort is made to expel it by blowing. Guinea pigs are not well suited to this method, as their small airway permits only the use of concentrated solutions.

Figure 2 illustrates the technic of intrabronchial instillation. The rabbit is completely immobilized in a standard box, the mouth is held open by a perforated tongue blade and a shortened no. 8 catheter is threaded through the larynx into the upper part of the trachea. Presence of the catheter in the trachea is established by expelling of air bubbles in a cup of water during respiration. Once the catheter is in position, the animal offers little or no resistance, and solutions are injected at the desired rate. A suitable method of catheterizing the tracheobronchial tree of the guinea pig was not devised.

For observation on the effects of ingestion, the animal's mouth is simply opened and measured amounts of anesthetic are introduced. Such apparent simplicity is deceptive, for the swallowing reflex may be quickly lost. If the solution is regurgitated into the nose or overflows into the trachea, results are highly irregular. Many

Fig. 2.—Position of rabbit for intrabronchial instillation of anesthetic solution.

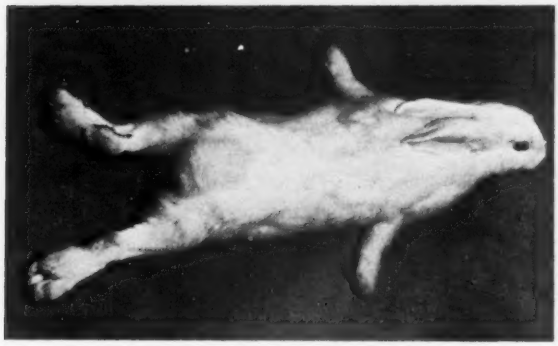

Fig. 3.—Mild reaction following intrabronchial injection of tetracaine.

guinea pigs were lost before this obvious fact was recognized, and deaths were at first attributed to "undue sensitivity" of certain animals. With dilute solutions, passage of a catheter into the upper portion of the esophagus is necessary. This group was fasted preliminarily for 48 hours so that stomach contents would not interfere with absorption.

All topical administrations were made over a period of three to four minutes.

MEASUREMENT OF ANIMAL RESPONSES

Minimum lethal doses were, of course, predicated on animal death. Toxic reactions short of death were, for purposes of this investigation, best demonstrated by gross changes in motor activity. These changes are definite, uniform and consistent, varying only in severity. With both cocaine and tetracaine the first visible evidence of reaction is incoordination, the animal being unable to control its extremities and move about normally. Figure 3 illustrates this stage in the rabbit. If the reaction is moderate, the animal falls on its side and lies in a state of extensor spasm (fig. 4). In severe reactions this spasm is accompanied with convulsions, which may terminate in death. There are certain differences between cocaine and tetracaine reactions which render them distinct from each other, but the basic pattern is similar.

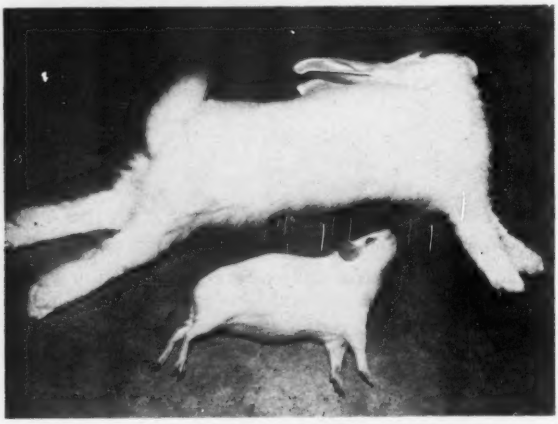

Fig. 4.—Moderate reaction in rabbit following intrabronchial injection of cocaine and in guinea pig following subcutaneous administration of tetracaine.

EXPERIMENTAL FINDINGS

Solutions Administered by Mouth.—Cocaine, 10 per Cent: Reactions, as shown in figure 4, were constant at 1.2 cc. per kilogram, and death occurred at 1.5 cc. per kilogram and above. Guinea pigs were slightly more sensitive, reacting at 0.8 to 1.1 cc. per kilogram and dying at 1 cc. per kilogram and above. All died at 1.2 cc. per kilogram.

Cocaine, 50 per Cent (guinea pigs only): Results were exactly the same proportionately as with cocaine, 10 per cent.

Tetracaine, 2 per Cent: Rabbits reacted within a range of 1.6 to 2.5 cc. per kilogram and died at 2.5 cc. per kilogram and above. Guinea pigs showed reactions at anywhere from 2 to 3 cc. per kilogram, with deaths starting to occur at 2.5 cc. per kilogram and being constant above 3.2 cc. per kilogram.

Reactions were varied as to time of onset, beginning anywhere from one-half to three hours after ingestion. Epinephrine, 1:1,000, from 1 to 3 minims per cubic centimeter, did not affect the above figures in the slightest.

The amounts of solution which these animals tolerate by ingestion before experiencing reactions are impressive, exceeding in 1 kilogram the maximum recommended dose for the human adult. Comparable doses in man would be expected invariably to cause death. It should be noted that neither concentration of solution nor the addition of epinephrine affected either the time of onset or the intensity of reactions.

Solutions Administered by Nose (rabbits only).—Cocaine, 10 per Cent: Reactions occurred at 0.4 to 0.6 cc. per kilogram, with deaths beginning at 0.6 cc. per kilogram. The addition of epinephrine, 2 (0.12 cc.) minims per cubic centimeter, protected against minimal lethal doses. Animals experienced moderate to severe reactions at 0.8 cc. per kilogram but did not die.

Cocaine, 50 per Cent: There were reactions at 0.08 to 0.12 cc. per kilogram, with deaths starting at 0.12 cc. per kilogram and being constant at 0.13 cc. per kilogram. Epinephrine, 1:1,000, from 1 minim to equal amounts, did not alter the response or time of onset. If anything, with it reactions seemed slightly severer.

Tetracaine, 2 per Cent: Reactions were mild at 0.2 cc. per kilogram, moderate at 0.25 to 0.3 cc. per kilogram and severe at 0.3 cc. per kilogram, with deaths starting here and being constant above this point. These reactions were characterized by their rapidity of onset near the minimum lethal dose. Although three to four minutes were arbitrarily taken to instil the solution, animals were already in spasm by the time instillation was completed. With cocaine several additional minutes elapsed before reactions were evident. The protective effect of epinephrine was marked. With 2 minims per cubic centimeter of epinephrine almost twice as much could be administered intranasally before death was caused. The invariably lethal dose, 0.35 cc. per kilogram, produced only a mild reaction if epinephrine was added. Death occurred at 0.6 cc. per kilogram.

The nasal airway of the guinea pig is so small that solutions are lost in exhalation before more than mild reactions occur, and results above this level are unreliable. Doses that produced mild reactions in rabbits had the same effect in the guinea pigs.

Comparison with oral administration reveals that absorption from the nose is much more rapid, and, hence, the tolerance is far less. In fact, the minimum lethal dose of cocaine by mouth is about two and one-half times and tetracaine about seven times greater than by nose. Epinephrine was significantly protective with tetracaine, 2 per cent, and cocaine, 10 per cent, but not at all with cocaine, 50 per cent.

Solutions Administered Intrabronchially (rabbits only).—Cocaine, 10 per Cent: There were no reactions at 0.05 cc. per kilogram and below. Reactions were mild to severe from 0.1 to 0.28 cc. per kilogram and death occurred at 0.27 cc. per kilogram and over. With the addition of epinephrine, 1:1,000, 1 to 2 minims per cubic centimeter, and slow administration over a three to four minute period, there was no change in intensity of reactions at any dose level. Epinephrine, in other words, was not protective.

Cocaine, 50 per Cent: Reactions were mild to moderate at 0.04 to 0.05 cc. per kilogram, with deaths at 0.06 cc. per kilogram. Epinephrine, from 1 minim to equal parts with cocaine, produced no visible changes in reaction features.

Tetracaine, 2 per Cent: There were no reactions at 0.1 cc. per kilogram. They were moderate to severe at 0.15 to 0.2 cc. per kilogram, and death took place at

0.22 cc. per kilogram and over. The addition of epinephrine was protective against a lethal dose, deaths taking place at 0.3 cc. per kilogram and above.

Tetracaine, 0.5 per Cent: When this dose was administered over the same period of time as the 2 per cent solution the reactions were identical.

The relatively greater sensitivity of the tracheobronchial tree, as compared to other mucous membranes, is clearly demonstrated. Also emphasized are the protective effect of epinephrine with tetracaine, the lack of it with cocaine and the failure of increasing dilution to decrease toxicity.

HALF COCAINE AND HALF TETRACAINE

Sollmann [3] stated that the efficiency of mixtures of the local anesthetics corresponds more or less to complete summation without any potentiation. These studies were corroborative. In intrabronchial administration of equal amounts of cocaine and tetracaine, half the amount of each required to produce a reaction produced the same response as a full dose of either when combined.

SUBCUTANEOUS INJECTION

Although this investigation is concerned with the toxicity of topical anesthetics topically applied, observations on the effects of subcutaneous injection were made for comparative purposes. These examinations proved important as a control measure, since there appears to be no agreement as to what constitutes minimum lethal doses in rabbits and guinea pigs. Hirschfelder and Bieter [4] charted the findings of 19 independent investigators, and the minimum lethal dose for cocaine in rabbits by subcutaneous administration varied from 50 to 200 mg. per kilogram. For guinea pigs, in 14 reports the range was from 20 to 80 mg. per kilogram. These variations are unaccountably wide. With tetracaine only one study on rabbits has apparently been recorded, that by Fussgänger and Schaumann [5] in 1931. They reported a minimum lethal dose of 20 mg. per kilogram. No estimates are to be found for the guinea pig. Results of the present investigation follow:

Rabbits.—Cocaine, 10 per Cent: Reactions occurred at 0.3 to 0.7 cc. per kilogram and deaths at 0.65 cc. per kilogram and above; 0.65 cc. is the equivalent of 65 mg., which is within but at the lower end of the range reported by Hirschfelder and Bieter.

Tetracaine, 2 per Cent: Reactions occurred over a wide range, from 0.15 to 0.9 cc. per kilogram, with deaths starting at 0.9 cc. per kilogram and over. This is the equivalent of 18 mg., which agrees closely with the findings of Fussgänger and Schaumann.

Guinea Pigs.—Cocaine, 10 per Cent: Reactions occurred between 0.15 and 0.3 cc. per kilogram, with death starting at 0.3 cc. per kilogram and being constant at 0.4 cc. per kilogram; 0.3 cc. contains 30 mg., which falls in the lower part of the minimum lethal death range reported by Hirschfelder and Bieter. Epinephrine was protective, no reactions taking place below 0.45 cc. per kilogram. Animals died with doses at 0.6 cc. per kilogram and above.

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3. Sollmann, T. H.: A Manual of Pharmacology and Its Applications to Therapeutics and Toxicology, ed. 7, Philadelphia, W. B. Saunders Company, 1948, p. 264.

4. Hirschfelder, A. D., and Bieter, R. N.: Local Anesthetics, Physiol. Rev. **12**:190, 1932.

5. Fussgänger, R., and Schaumann, O.: Über ein neues Lokalanästhetikum der Novokainreihe (Pantokain), Arch. f. Exper. Path. u. Pharmakol. **160**:53, 1931.

Tetracaine, 2 per Cent: As in rabbits, there was a wide range of reaction, from 0.1 to 1.4 cc. per kilogram. Deaths began at 1.3 cc. per kilogram and were constant above 1.5 cc. per kilogram. Epinephrine exerted a remarkably strong protective action. With 2 minims per cubic centimeter reactions did not begin until administration of 4 cc. per kilogram, and there were no deaths with doses under 4.5 cc. per kilogram. In other words, the animals tolerated three times as much tetracaine if epinephrine was added.

It is evident from these findings that the minimum lethal doses of cocaine and tetracaine by subcutaneous injection are not the same as those by topical administration, and therefore it follows that experimental determinations of toxicity by injection are not true indications of toxicity by topical application.

Summary of Comparative Toxicities of Cocaine and Tetracaine, in Milligram per Kilogram, in the Rabbit

	Cocaine, 10%		Tetracaine, 2%		Ratio Tetracaine to Cocaine
	Reactions	MLD*	Reactions	MLD*	
Mode of administration					
Ingestion					
Drugs alone	120 to 155	150	30 to 60	50	3:1
With epinephrine	Same		Same		
Nasal instillation					
Drugs alone	50 to 60	60	3 to 6	7	8½:1
With epinephrine	80+	Not determined	5 to 10	10	...
Tracheobronchial instillation					
Drugs alone	10 to 28	27	2 to 4	4½	6:1
With epinephrine	Same		? to 5	6	...
Subcutaneous injection					
Drugs alone	30 to 70	65	3 to 19	18	3½:1

* Minimum lethal dose.

TIME

This investigation is not primarily concerned with time over which anesthetics are administered as a factor in producing reactions, since another variable is thereby introduced. The matter of time is the subject of a separate study. In these experiments three to four minutes were arbitrarily selected as the period within which the anesthetic solutions would be administered. The time being constant minimized it as a source of error in establishing relative speed of absorption by various mucous membranes. However, the importance of the time factor cannot be overemphasized. For example, 0.2 cc. per kilogram of tetracaine 2 per cent instilled intrabronchially in three to four minutes produced a reaction but was not lethal. The same amount administered in 15 seconds invariably caused death. Both the 10 and the 50 per cent solution of cocaine caused only moderate reactions when instilled in three to four minutes but were lethal when instilled in less than 15 seconds.

COMMENT

Rate of Absorption by Mucous Membranes.—These experiments demonstrate a great difference in the absorptive abilities of various mucous membranes in the rabbit, and though the experimental findings cannot be unqualifiedly applied to man, it is probable that there is at least some similarity. The tracheobronchial tree proved to be the most sensitive, undoubtedly because of the unavoidable

entrance of solution into the alveoli. The nose tolerated twice as much cocaine and one and a third times as much tetracaine as the tracheobronchial tree. Uptake by mouth, pharynx, esophagus and stomach was comparatively slow, the animals tolerating five times as much cocaine and 10 times as much tetracaine by this route as by the route of the lower respiratory tract. Greater tolerance to tetracaine here as compared to cocaine is explained by the greater speed with which it is known to be detoxicated in the rabbit. Absorption from the highly vascular nasal mucous membrane, however, appears to be so rapid that a high blood concentration was quickly attained and the animal overwhelmed.

The rate of absorption of the tested anesthetic solutions and subsequent development of toxic manifestations followed a definite and consistent pattern for each mucous membrane. Reactions could be virtually pinpointed at will without loss of an animal. Only as the minimum lethal dose was approached did overlapping occur. Each mucous membrane has its own tolerance, and to make the mistake, for example, of instilling intrabronchially an anesthetic solution calculated to produce only a moderate reaction when administered intranasally results in prompt death. These variations in mucosal tolerance suggest that the word "topical" without reference to specific mucous membranes is as meaningless as the unqualified term "injection."

Concentration of Solution and Toxicity.—That dilution of parenterally administered local anesthetics decreases their toxicity is well known. It has apparently been assumed that the same is true of anesthetics topically applied, for otolaryngological textbooks and papers have continued to insist on the danger of concentrated solutions. In recent years this warning has taken the form of asserting that toxicity rises with increasing concentration of a topical anesthetic in geometric and not arithmetical progression. In other words, a 2 per cent solution of tetracaine, for example, is not twice but four times as toxic as a 1 per cent solution. Opposed to this opinion is a minority which has held that cocaine "mud" is just as safe or safer than the dilute solutions because it is applied to a limited area and absorbed more slowly over a small surface. Accidents have occurred with both technics, and in 1924 the Mayer Committee[6] of the American Medical Association recommended specific doses which tended to outlaw concentrates. Here the matter has rested.

In these studies, tetracaine, 0.5 and 2 per cent, was administered intrabronchially to rabbits; cocaine, 10 and 50 per cent, intranasally, intrabronchially and by mouth to rabbits, and cocaine, 10 and 50 per cent, by mouth to guinea pigs. Responses in the lesser and greater concentrations were identical, that is, with time of administration constant, the total dose in milligrams of drug was the only determining factor in reactions. Dilution did not in the least decrease toxicity. Theoretically, these seemingly contradictory findings are not at all inconsistent with the accepted concept of lessening toxicity of injected anesthetics by dilution. When an anesthetic solution is injected subcutaneously, it pools at that point, and absorption is limited to that part of it directly in contact with tissue. The greater the dilution, the slower must be the absorption. With topical administration no such situation obtains, as there is no opportunity for accumulation. Whatever solution is instilled diffuses rapidly and contacts absorbing surfaces at once. Dilute

6. Mayer, E.: The Toxic Effects Following the Use of Local Anesthetics, J. A. M. A. **82**:876 (March 15) 1924.

solutions simply spread out over a greater area but are just as available for immediate absorption. From a clinical standpoint, the principle trouble with concentrated solutions would appear to be the tendency of the physician to forget that the solution he is using so freely is concentrated.

The Effect of Epinephrine.—That epinephrine is effective in decreasing toxicity of injected local anesthetics was first demonstrated by Braun [7] in 1903. In these studies the addition of epinephrine, 1:1,000, to tetracaine, 2 per cent, tripled the minimum lethal doses when injected subcutaneously. However, this same dramatic protective effect was not noted on topical administration. Epinephrine was completely ineffective with solutions administered by mouth and in all tests with cocaine, 50 per cent. Administered via the nose it afforded significant protection against both tetracaine, 2 per cent, and cocaine, 10 per cent (see table). The effect of epinephrine in the tracheobronchial tree was complicated by the element of time. For example, 0.15 cc. per kilogram of cocaine, 10 per cent, instilled in 15 seconds produced no more than a moderate reaction. With epinephrine, 1:1,000, 2 minims per cubic centimeter of anesthetic, in administration in 15 seconds, this dose became lethal. A dose of 0.1 cc. per kilogram of 2 per cent tetracaine solution instilled in 15 seconds never caused more than a moderate reaction, that is, extensor spasm without convulsions. With epinephrine, and administration in the same period, this reaction was accompanied with severe convulsions. If these same solutions were administered over three to four minutes, there was a definite protective effect against tetracaine and none against cocaine, but at least in the latter case there was no potentiation of toxicity. Ross [8] and Gold [9] found that epinephrine by intravenous injection enhanced the toxicity of cocaine for cats, and the results of these studies suggest that epinephrine increases toxicity of both cocaine and tetracaine in rabbits when given sufficiently rapidly intrabronchially to approach the speed of intravenous administration. The effect of very small amounts of epinephrine was not tested.

Relative Toxicities of Cocaine and Tetracaine.—Based on the investigations of Fussgänger and Schaumann [7] with rabbits, the toxicity of tetracaine by subcutaneous injection is considered to be two and one half to three times that of cocaine. These studies are corroborative. This, however, is by no means an accurate representation of relative toxicities when the same drugs are applied topically. A glance at the table of comparative toxicities discloses a range in the toxicity ratios of tetracaine to cocaine of from 3:1 by mouth to 8½:1 by nose to 6:1 by tracheobronchial tree. Which ratio applies most closely in topical administration to man, and it does not appear to be the subcutaneous, which is the generally accepted standard, is a matter of conjecture. From the standpoint of the otolaryngologist, the ratio which merits the greatest consideration is the intrabronchial. Administration here is to a mucous membrane, simulating otolaryngological application, yet

7. Braun, H.: Über den Einfluss der Vitalität der Gewebe auf die örtlichen und allgemeinen Giftwirkungen localanästhesirender Mittel und über die Bedeutung der Adrenalins fur die Localanästhesis, Arch. f. klin. Chir. **69**:541, 1903.

8. Ross, J.: Toxicity of Cocaine as Influenced by Rates of Absorption and Presence of Adrenalin, J. Lab. & Clin. Med. **8**:657, 1923.

9. Gold, H., cited by Mayer, E.: The Toxic Effects Following the Use of Local Anesthetics, J. A. M. A. **82**:876 (March 15) 1924.

absorption is so rapid as to permit the appearance of toxic manifestations before various extraneous factors can operate. If the ratio of toxicity of tetracaine to cocaine is, for over-all topical purposes, considered to be 6:1, which is the ratio by intrabronchial administration, then 1 cc. of cocaine, 6 per cent, will be held equal in toxicity to 1 cc. of tetracaine, 1 per cent.

CONCLUSIONS AND SUMMARY

The absolute and relative toxicities of cocaine and tetracaine by topical application were determined in the rabbit and guinea pig.

A considerable variation in the rate of absorption by different mucous membranes was found, thereby suggesting that the word "topical" by itself is no more descriptive than the unqualified term "injection."

Total dosage of the drugs in milligrams was the chief factor in determining the severity of toxic reactions. Dilution did not decrease toxicity.

The effect of epinephrine in conjunction with the local anesthetics was varied and was, for the most part, favorable, but not so much as in subcutaneous injection.

The time over which a drug was administered played a role in its toxicity.

Toxic doses of cocaine and tetracaine by subcutaneous administration did not accurately parallel those by topical administration, and it is suggested that the toxicity of topical anesthetics be determined by topical and not by injection methods.

3875 Wilshire Boulevard.

Winthrop-Stearns, Inc., furnished a complete bibliography on tetracaine toxicity.

STREPTOMYCIN IN OTOLARYNGEAL TUBERCULOSIS

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APPROXIMATELY one year ago, a paper [1] was published describing 57 tuberculous patients with otolaryngeal complications treated at the Fitzsimons General Hospital with streptomycin. Studies had been made with these patients since July 1946, when Fitzsimons General Hospital became a participating hospital in the federal services joint program of streptomycin research. The cases studied during the past year do not differ materially in qualitative results of therapy from those studied during the latter part of the previous period, but they do add quantitative support to the striking results experienced with this new therapeutic agent.

A detailed analysis of these cases will not be given, since it is generally well known that low dosages of streptomycin are just as efficacious as the originally used high dosages and that administration through the intramuscular route is preferable to local therapy for otolaryngeal lesions. There now appear to be prospects of several new methods in the treatment of tuberculosis, and these, together with the specific treatment of otolaryngeal lesions, will be reviewed along with some of the literature on these subjects.

It appears that some physicians have directed treatment merely at clearing up the local otolaryngeal lesion without integrating this therapy with the total treatment of the tuberculous patient, so that it is essential to emphasize this error. It is well known that unhealed tuberculous laryngeal and tracheobronchial lesions are usually associated with unhealed pulmonary lesions. Either of the foci, endoscopic or pulmonary, tend to have an unfavorable effect on the healing of the other, and either lesion, when active, makes the patient a potential spreader of the disease to his own system (endogenous spread) or a carrier to others (exogenous spread). It is proposed, then, not only to discuss the newer antibiotic and chemotherapeutic agents as to their effect on the local otolaryngeal lesions but to correlate these effects with the treatment of the tuberculous patient as a whole. It is proposed, also, to elaborate briefly on the public health aspects of the lesions themselves and on the public health aspects of the newer therapeutic agents. In this connection it is worth while to mention that these newer agents are being used to prepare the patient for earlier collapse and excisional surgery.[2] This use, of itself, is a significant advance in the treatment of tuberculosis and in public health control.

From the Eye, Ear, Nose and Throat Service, Fitzsimons General Hospital, Denver.

1. Vadala, A. J.: Streptomycin in Otolaryngic Tuberculosis, Laryngoscope **59**:156-168 (Feb.) 1949.

2. Forsee, J. H.: Streptomycin in the Surgical Therapy of Pulmonary Tuberculosis, J. Internat. Coll. Surgeons **12**:707-727 (Sept.-Oct.) 1949. Beattie, J. B., Jr., and Blades, B.: Use of Streptomycin in Surgical Patients, J. A. M. A. **139**:902-906 (April 2) 1949

If tuberculosis of the larynx occurred as a local lesion only, that is, not in association with tuberculosis elsewhere in the body, then it would be safe to say that with the present methods of therapy this condition could soon be wiped out. While most of these lesions clear in a short time under proper therapy, the treatment is likely to prove inadequate for associated pulmonary lesions, and the patient may have been started well on his way toward development of a resistant organism. It is of little benefit to clear a local lesion when the patient is in jeopardy of spread of his general disease and also of recurrence of the local lesion that has been arrested.

INCIDENCE AND CONTROL OF OTOLARYNGEAL TUBERCULOSIS

In a previous paper [1] it was stated that the incidence of laryngeal tuberculosis in relation to tuberculosis in general appeared to be on the decrease, and some of the possible reasons for this statement were postulated. Looper and Lyon [3] have recently discussed this matter in detail and have submitted considerable supporting statistics. Thomas Parran,[4] in 1944, when he was Surgeon General of the United States Public Health Service, stated that the estimated tuberculous population in the United States in 1938 was 1,500,000. Taking Myerson's [5] figure of 10.6 per cent as the incidence of laryngeal tuberculosis among tuberculous patients in general would give an estimated figure of 159,000 persons with laryngeal tuberculosis in the United States. Admittedly, this figure is too high. Myerson's figures were based on a survey of 10,657 tuberculous patients, but these were patients being admitted to sanatorium care, and the incidence of laryngeal complications in this group would be higher than in tuberculous persons in the population at large. It will be seen that even with this higher figure the task of clearing up laryngeal tuberculosis would not be difficult, provided that the tuberculous patient as a whole did not have to be considered. There are 4,262 certified otolaryngologists now in the United States,[6] and in computation with the above figures, each specialist would be required to care for about 37 patients.

What are the factors that have retarded a more complete control of tuberculosis? Stewart [7] states that there have been 2,100 papers written between 1916 and 1942 dealing with general measures to reduce tuberculosis among the general population. Perhaps some of the most prominent of the factors are as follows:

1. Lack of fund raising or appropriations to carry out the control measures
2. Lack of a nationally coordinated program to survey all the people periodically to detect such cases
3. Insufficiency of beds to care for the tuberculous patient
4. Inadequate control of known cases of tuberculosis
5. Lack of both popular and official full support for an "all out" program

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3. Looper, E. A., and Lyon, I. B.: Laryngeal Tuberculosis, Ann. Otol., Rhin. & Laryng. **57**:754-768 (Sept.) 1948.

4. Parran, T.: Outlook for Tuberculosis Control in Civilian Population, Am. Rev. Tuberc. **50**:365-374 (Nov.) 1944.

5. Myerson, M. C.: Tuberculosis of the Ear, Nose, and Throat, Including the Larynx, the Trachea, and the Bronchi, Springfield, Charles C Thomas, Publisher, 1944.

6. Approved Examining Boards in Medical Specialties, editorial, J. A. M. A. **141**:63-64 (Sept. 3) 1949.

7. Stewart, C. A.: Symposium on Tuberculosis in Institutions: Tuberculosis Control Programs, Dis. of Chest **11**:298-303 (July-Aug.) 1945.

6. Lack of a known effective bacteriostatic or bactericidal agent for the tubercle bacilli. To elucidate the above points, two papers will be drawn on freely.[8]

Factor 1: There are approximately 95,000 hospital beds available for tuberculous patients, so that with Parran's estimate of the number of cases of tuberculosis, only about 6 per cent of such patients have the advantage of hospitalization at any one time. The investment in tuberculosis institutions amounts to $300,000,000, and the cost of hospitalizing tuberculous patients in 1940 was over $80,000,000. During 1940 the people of the United States spent approximately $475,000,000 for chewing gum, cigars, cigarets and ice cream. In 1945 the estimated cost of construction of a hospital bed was $6,000 to $7,000 and the cost of maintenance from $6.00 to $7.00 a day for each patient.

Factor 2: Piecemeal screening by local communities and pilot surveys of some of the large cities under national auspices have been done, but no "all out" attack for all the people at any one time has been accomplished.

Factor 3: This point has been considered under factor 1.

Factor 4: Physicians hesitate to and often do not report their private patients to the boards of health. Directors of boards of health hold appointive positions and hesitate to stir up opposition by enforcing laws. Known cases of tuberculosis are inadequately controlled, owing to lack of funds. Tuberculosis patients who know their condition is active take positions endangering others, perhaps because of lack of proper instruction or moral suasion or because of economic reasons. A case has been cited in the literature of a school bus driver who infected 30 children. Many times the person with early tuberculosis exposes others because he is not aware of his disease. At Fitzsimons General Hospital there have been as many as five admissions in one week of tuberculous food handlers and cooks.

Factor 5: It is probable that lack of enthusiasm on the part of the public has been largely due to the feeling that there was not available an agent of therapeutic efficiency. There has even been some opposition manifested toward mass roentgenography of the chest. The person suspecting that he may possibly have tuberculosis has, no doubt, visualized the possibility as being as undesirable as a lifetime prison sentence or a diagnosis of incurable cancer. A strong reason for indifference by large communities has probably been the feeling that since no determined national effort was in progress any gains accomplished locally could soon be overrun by influx from neighboring communities and the populace at large.

Public officials object to a program for clearing tuberculosis on the grounds of expense. It should be emphasized that tuberculosis is a slowly spreading epidemic disease, that it is highly expensive and that the people pay the bill. During the last few years, while the mortality rate for tuberculosis has been going steadily down, it has been estimated that the morbidity rate has been slowly creeping up. It has been shown by surveys in large cities that every dollar spent in finding early cases pays several hundred per cent savings to the community from future hospitaliza-

8. Laws, J. W.: Legislative Implications of Adequate Tuberculosis Control, Dis. of Chest **14**:121-131 (Jan.-Feb.) 1948. Nimitz, H. J.: The Problem of Tuberculosis in the Average Community, Dis. of Chest **10**:528-538 (Nov.-Dec.) 1944.

tion costs. The National Tuberculosis Association has estimated that the economic loss and cost of care and treatment of each death from tuberculosis is $10,000.

Factor 6: The newer therapeutic agents for the treatment of tuberculosis have rightfully been played down to the press and the public in order to avoid the consequences that follow premature announcements of this nature. It will be some years before adequate investigation of these agents has been made and before the results of the BCG vaccination are known. When these investigations have been accomplished, if the results are as good as they now are promising, these agents will be powerful in the appeal for more intensive action.

Parran [4] states that in the past 40 years the death rate from tuberculosis in the United States has been halved and then halved again. He also states, "It is a common experience in public health work that frontal attack on a widespread disease produces dramatic results in the initial phase, followed by a lag as volume of infection diminishes." It would appear that we are now not far from the time for another frontal attack phase.

BIOLOGICAL AND CHEMOTHERAPEUTIC AGENTS FOR TREATMENT OF TUBERCULOSIS

In 1944 Schatz and associates [9] announced that they had isolated streptomycin from Actinomyces griseus. After some experience with this agent in the therapy of tuberculosis it became apparent, as expressed by Tucker,[10] that "The course of the Good Ship Streptomycin lies between the Scylla of toxicity and the Charybdis of bacterial resistance." Table 1 is a listing of the different regimens of therapy that have been investigated or are currently under investigation at this hospital. The purpose of all these regimens has been to determine the most effective drug or combination of drugs as well as the optimum dosage and dosage schedules. These regimens should be adequate for treatment of the tuberculosis but should delay or eliminate the emergence of resistant organisms and delay or eliminate toxic manifestations.

The comparison of different dosage regimens investigated by the federal service group was reported by Tucker,[10] and those regimens specifically investigated at Fitzsimons General Hospital, up to and inclusive of regimen 9, were reported by Deyke and associates [11] and Tempel and Dye.[12] Particular attention is directed to regimens 14 and 15, the intermittent combined therapy schedules that received original investigation at this hospital. While the table does not show the results of emergence of resistance or therapeutic results, it can be said that these two regimens, 14 and 15, have been the most successful of any so far studied from both standpoints. The regimens starting with number 16 and up have not been investigated sufficiently at this time to warrant any definite conclusions. More than

9. Schatz, A.; Bugie, E., and Waksman, S. H.: Streptomycin: A Substance Exhibiting Antibiotic Activity Against Gram Negative and Gram Positive Bacteria, Proc. Soc. Exper. Biol. & Med. **55**:66-69 (Jan.) 1944.

10. Tucker, W. B.: Evaluation of Streptomycin Regimens in the Treatment of Tuberculosis, Am. Rev. Tuberc. **60**:715-754 (Dec.) 1949.

11. Deyke, V. F.; Fisher, M. W.; James, L. A., and Sides, L. J.: Intermittent Dosage Schedules of Streptomycin with Resultant Prolonged Sensitivity of M. Tuberculosis, Ann. Int. Med. **30**:619-634 (March) 1949.

12. Tempel, C. W., and Dye, W. E.: Selecting the Streptomycin Regimen for Patients with Pulmonary Tuberculosis with Special Reference to the Intermittent Dosage Schedule, Dis. of Chest **16**:704-713 (Dec.) 1949.

100 patients have completed therapy under regimens 14 and 15, but the project on these two regimens has not been completed. At the present time, only one patient has shown resistance, and none have had depressed labyrinthine function.

Further comment is necessary to explain the basis on which the intermittent combined therapy schedule was devised. The combined therapy principle is based on the work of Demerec.[13] He asserted that the most effective way of preventing the emergence of resistant organisms was by using simultaneously two agents that were independently active against the organisms. Thus only organisms which can withstand the two drugs at the same time can emerge resistant. For instance, if bacteria are resistant to one therapeutic agent to the frequency of 10^{-6} and to

TABLE 1.—*Regimens for Treatment of Tuberculosis*

Regimen No.	Starting Date	Drug	Dosage Schedule
x	Prior to April 1946	....	Irregular (other regimens)
1	April 1946	SM	2 Gm. (1 Gm. b.i.d.) 120 days
2	May 1947	SM	1 Gm. (single dose) 120 days
3	May 1947	SM	1 Gm. (single dose) 90 days
4	April 1948	SM	1 Gm. (single dose) 42 days
5	April 1948	SM	1 Gm. (single dose) 28 days
6	April 1948	SM	½ Gm. (single dose) 28 days
7	January 1949	DHSM	2 Gm. q.3d. 30 days
8	May 1948	SM	2 Gm. q.3d. 120 days
9	May 1948	SM	1 Gm. q.3d. 120 days
10	February 1949	PAS	12 Gm. (3 Gm. q.i.d.) 120 days
11	May 1949	PAS SM	12 Gm./d. plus 2 Gm. q.3d. 120 days
12	May 1949	PAS SM	12 Gm./d. plus 1 Gm. q.3d. 120 days
13	September 1948	SM	1 Gm./d. 28 days; rest, 42 days; 1 Gm./d. 28 days
14	November 1949	PAS SM	12 Gm. q.3d. plus 2 Gm. q.3d. 120 days
15	November 1949	PAS SM	12 Gm. q.3d. plus 1 Gm. q.3d. 120 days
16	December 1949	TB-1	200 mg./d. 120 days
17	December 1949	TB-1 SM	200 mg./d. plus 1 Gm. 120 days
18	December 1949	TB-1 SM	200 mg. q.3d. plus 2 Gm. q.3d. 120 days
19	December 1949	TB-1 SM	200 mg. q.3d. 1 Gm. q.3d. 120 days

SM indicates streptomycin; DHSM, dihydrostreptomycin; PAS, paraaminosalicylic acid, and TB-1, Tibione.®

another to the frequency of 10^{-5}, they will be resistant to the two drugs used together to the frequency of 10^{-11}. Karlson and associates[14] and Delande and associates[15] have also reported that simultaneous administration of different tuberculostatic drugs appeared to delay the emergence of resistance.

13. Demerec, M.: Origin of Bacterial Resistance to Antibiotics, J. Bact. **56**:63-74 (July) 1948.

14. Karlson, A. G.; Pfeutze, K. H.; Carr, D. T.; Feldman, W. H., and Hinshaw, H. C.: The Effect of Combined Therapy with Streptomycin, Para-aminosalicylic Acid and Promin on the Emergence of Streptomycin-Resistant Strains of Tubercle Bacilli; A Preliminary Report, Proc. Staff Meet., Mayo Clinic **24**:85-88 (Feb. 16) 1949.

15. Delande, A.; Karlson, A. G.; Carr, D. T.; Feldman, W. H., and Pfeutze, K. H.: Increase in Resistance of Tubercle Bacilli to Sodium P-Aminosalicylic Acid: Observations on Cultures Isolated From Patients During Chemotherapy, Proc. Staff Meet., Mayo Clinic **24**: 341-345 (June 22) 1949.

Still another effect of the combined administration of therapeutic agents, besides the delayed emergence of resistance, is the additive therapeutic effect. Thus the effect of two antibiotics in combination is greater than can be expected from the optimum therapeutic dosage of either agent when it is used alone. This observation has been reported by Karlson and Feldman [16] in experiments with guinea pigs, by Youmans and Osborne [17] with mice and by Bloch and associates [18] with both in vivo and in vitro methods. An interesting in vivo experiment was performed by Rees and Robson [19] in which they produced tuberculous lesions on the eyes of rabbits by injecting 300 tubercle bacilli intracorneally. After the lesions were well formed, Rees and Robson injected streptomycin into the vitreous of some of the eyes, paraaminosalicylic acid into some and streptomycin and paraaminosalicylic acid in combination into others; other eyes were used as controls. They were able, in this way, to demonstrate a visible additive effect of streptomycin and paraaminosalicylic acid in vivo.

The investigation of intermittent regimens, as pioneered at Fitzsimons General Hospital, was started in May 1948. The therapy is administered every three days instead of daily. The rationale, as stated by Hughes,[20] was based on three observations: (1) that the tubercle bacilli reproduce in the test tube approximately every three days [21]; (2) that streptomycin is most actively bacteriostatic at the time of fission,[22] and (3) that streptomycin in guinea pigs is capable of exerting a level of sustained therapeutic effectiveness when administered intermittently.[23]

One other regimen studied at this hospital and not shown in table 1 is an interrupted regimen for the treatment of meningeal tuberculosis, miliary tuberculosis and the two conditions occurring in the same patient.[24] Approximately 40 patients were treated, about half on a continuous daily schedule and about half on an interrupted schedule. The interrupted schedule consisted of 28 days of continuous daily treatment followed by 28 days of rest. The patients on the interrupted schedule have fared far better than those on the continuous schedule, owing to the delay in emergence of resistance.

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16. Karlson, A. G., and Feldman, W. H.: The Effect of Combined Therapy with Streptomycin and Para-Aminosalicylic Acid (PAS) on Experimental Tuberculosis in Guinea Pigs, Proc. Staff Meet., Mayo Clin. **24**:510-515 (Sept. 28) 1949.

17. Youmans, A. S., and Osborne, R. R.: Combined Effect of Streptomycin and Para-Aminosalicylic Acid on Experimental Tuberculosis in Mice, Journal-Lancet. **67**:403-404 (Nov.) 1947.

18. Bloch, R. G.; Kirsten, V.; Ebert, R. H., and Gomori, G.: The Effect of Streptomycin, Para-Aminosalicylic Acid (PAS) and Their Combination on the Tubercle Bacillus in Vitro and in Vivo, Am. Rev. Tuberc. **59**:554-561 (May) 1949.

19. Rees, R. J. W., and Robson, J. M.: Additive Effect of Streptomycin and P-Aminosalicylic Acid in Experimental Tuberculous Infection, Nature **164**:351 (Aug. 27) 1949.

20. Hughes, F. J., Jr.: Personal communication to the author.

21. Youmans, G. P.: A Method for the Determination of the Culture Cycle and Growth Rate of Virulent Human Type Tubercle Bacilli, J. Bact. **51**:703-710 (June) 1946.

22. Middlebrook, G., and Yegian, D.: Certain Effects of Streptomycin on Mycobacterium in vitro, Am. Rev. Tuberc. **54**:553-558 (Dec.) 1946.

23. Carper, H. J., and Conn, M. L.: Remote Sustained Threshold Therapeutic Action of Streptomycin in Tuberculosis, Science **106**:446-447 (Nov. 7) 1947. Feldman, W. H.; Hinshaw, H. C., and Karlson, A. G.: Frequency of Administration of Streptomycin, Am. Rev. Tuberc. **55**:435-443 (May) 1947.

24. Towbin, M. N.: Personal communication to the author.

The above examples of the use of streptomycin and paraaminosalicylic acid in combination for additive effect and the principle of intermittent and interrupted regimens to delay toxicity and resistant organisms portend the possibilities which can reasonably be expected as some of the newer therapeutic agents become more thoroughly investigated. These agents of promising value are being announced with such rapidity that clinical investigation is far in arrears. This situation is not surprising when the chronicity and protean nature of tuberculosis is taken into account and, also, that streptomycin, which has been investigated six years, still presents unanswered questions. A few of the newer tuberculostatic agents other than streptomycin will be described briefly.

Paraaminosalicylic acid is rapidly absorbed by the oral route and rapidly excreted. It is detoxified in the body by acetylation, presumably by the liver. The dose, as used at this hospital, has been 12 Gm. daily, given in four divided doses, which results in blood levels of about 1 mg. per cent.[25] This level has been found to inhibit tubercle bacilli in vitro. The drug can also be given intravenously in emergency cases such as tuberculous meningitis.

Tibione® [26] is 4-acetylaminobenzaldehyde thiosemicarbazone. It was developed in Germany in 1946 and is supplied in 25 and 50 mg. tablets for oral use. It is rapidly absorbed from the gastrointestinal tract and more slowly excreted in the urine in about 15 per cent of the original amount. It is 10,000 times less tuberculostatic in vitro than paraaminosalicylic acid but is as effective in vivo in 50 times smaller dose. It is thought to exert a competitive inhibition phenomenon on the metabolites necessary for the multiplication of the tubercle bacilli. The usual adult dose is 2 mg. per kilogram of body weight perorally daily.

Neomycin [27] is more active against pathogenic mycobacteria than other present antibiotics. It is just as active against streptomycin-resistant as streptomycin-sensitive mycobacteria. This drug does not allow as rapid development of resistance of the mycobacteria as streptomycin. Pathogenic mycobacteria are inhibited by 0.2 to 1.0 units per milliliter. In the experimental animal it is effective against pathogenic mycobacteria in one twentieth to one fiftieth of the toxic dose. No reports have yet been made of its clinical use in tuberculosis.

Terramycin has recently been isolated from a new actinomycete, Streptomyces rimosus, at the laboratories of Charles Pfizer & Co., Inc., by Finlay and associates.[28] It is reported as being effective against a wide range of both gram-negative and gram-positive organisms. It has not as yet been tried clinically against tuberculosis.

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25. Dye, W. E., and Weiser, O. L.: Para-Aminosalicylic Acid Blood Levels, Bull. U. S. Army M. Dept. **9**:1053-1055 (Dec.) 1949.

26. A New Drug in the Treatment of Tuberculosis, editorial, J. A. M. A. **142**:342-343 (Feb. 4) 1950. Benisch, R.; Meitzsch, F., and Schmidt, H.: Chemical Studies on Thiosemicarbazones with Particular Reference to Antituberculous Activity, Am. Rev. Tuberc. **61**:1-7 (Jan.) 1950. Domagk, G.: Investigations on the Antituberculous Activity of the Thiosemicarbazones in Vitro and in Vivo, Am. Rev. Tuberc. **61**:8-19 (Jan.) 1950. Mertens, A., and Bunze, R.: The Present Status of the Chemotherapy of Tuberculosis with Conteben, A Substance of the Thiosemicarbazone Series, Am. Rev. Tuberc. **61**:20-38 (Jan.) 1950.

27. Waksman, S. A.; Hutchison, D., and Katz, E.: Neomycin Activity upon Mycobacterium Tuberculosis and Other Mycobacteria, Am. Rev. Tuberc. **60**:78-89 (July) 1949.

28. Finlay, A. C., and others: Terramycin, A New Antibiotic, Science **111**:85 (Jan. 27) 1950.

A crystalline extract from the lichen Ramalina reticulata is an antibiotic substance isolated in a cooperative study by the Tuberculous Control Division, United States Public Health Service, and the Rockefeller Institute for Medical Research.[29] In vitro tests have shown tubercle bacilli growth to be completely inhibited in concentrations of 1 to 50,000, and inhibition is noticeable in concentrations as low as 1 to 2,000,000. The substance has been effective in suppressing tubercle bacilli growth in guinea pigs, but I have seen no report on its clinical trial.

TUBERCULOUS LARYNGITIS

Figi and associates [30] were probably the first to report on a patient with laryngeal tuberculosis treated with streptomycin. The patient was given 1 Gm. of streptomycin intramuscularly each 24 hours, and this dose was supplemented with streptomycin aerosol. There was an improvement in 10 days and a 75 per cent rate of improvement in 24 days; in six weeks the patient was considered cured. Greif and Gould [31] reported six cases of tuberculous laryngitis, with healing in four and improvement in two with streptomycin treatment varying from 10 days to about two months. They used both nebulization and the intramuscular route for administration of the drug and believed tentatively, that a combination of the two afforded the best results.

Black and Bogen [32] treated 34 patients with laryngeal tuberculosis with streptomycin and had superior results with the intramuscular administration in comparison to that of the aerosol. Withers [33] cured 10 of 12 patients with tuberculosis of the larynx, the condition in the other two being markedly improved on a regimen of 1 Gm. of streptomycin daily for 90 days. He stressed the unfavorable effect of tuberculosis of the larynx on the associated pulmonary tuberculosis, owing to odynophagia and, consequently, poor nutrition.

O'Keefe [34] reported 44 cases of tuberculous tracheobronchitis and 27 cases of tuberculous laryngitis in which streptomycin treatment was used. Ten of the patients with tracheobronchitis and five of those with laryngitis were given only streptomycin aerosol, with success in one in each subgroup. The remaining patients received 1 or 2 Gm. of streptomycin daily by the intramuscular route for 90 to 120 days. Only four of the 34 patients with tracheobronchitis and two of the 22 with laryngitis failed to improve with this treatment.

Margulies [35] compared results of various small intramuscular doses of streptomycin in 26 patients with tuberculous laryngitis. All patients had associated far

29. Weber, F. J.: Progress in Tuberculosis Control in the United States, Dis. of Chest **14**:243-253 (March-April) 1948.

30. Figi, F. A.; Hinshaw, H. C., and Feldman, W. H.: Treatment of Tuberculosis of the Larynx with Streptomycin: Report of Case, Proc. Staff Meet., Mayo Clin. **21**:127-130 (March 20) 1946.

31. Greif, J. L., and Gould, W. J.: Tuberculosis of Ear, Nose, and Throat Treated with Streptomycin, Arch. Otolaryng. **48**:209-220 (Aug.) 1948.

32. Black, M., and Bogen, E.: Streptomycin in Tuberculous Laryngitis, Am. Rev. Tuberc. **56**:405-407 (Nov.) 1947.

33. Withers, B. T.: Streptomycin in Treatment of Laryngeal Tuberculosis, Ann. Otol., Rhin. & Laryng. **57**:769-783 (Sept.) 1948.

34. O'Keefe, J. J.: Tuberculous Laryngitis and Tracheobronchitis, Ann. Otol., Rhin. & Laryng. **58**:441-447 (June) 1949.

35. Margulies, B.: Streptomycin in Tuberculosis of the Larynx, Dis. of Chest **15**:432-435 (April) 1949.

advanced pulmonary lesions and sputum positive for the organism. They were all treated for three months. Following are the doses and the results: Five patients received 0.1 Gm. daily, with improvement in one; eight, 0.2 Gm. daily, with improvement in 75 per cent; five, 0.5 Gm. daily, with improvement in 80 per cent, and eight, 1 Gm. weekly, with improvement in 62 per cent. He states that, in general, about 70 per cent of the patients treated with 1 to 2 Gm. daily improved.

Table 2 shows the results of streptomycin treatment in otolaryngeal cases at this hospital. The condition in about three fourths of the cases was considered as cleared, in one-fifth as improved and in one-tenth as unchanged. In general, it can be said that in the majority of these cases clearance occurred in about two months with intramuscular administration of streptomycin in dosages between 0.5 to 2 Gm., either intermittent or daily. The regimens together with the dates they were started are shown in table 1. The lack of cure in the cases of laryngeal tuberculosis is the result of either emergence of resistance or associated cavitary lesions that did not improve with proper therapy. It is believed that as more of

TABLE 2.—*Tuberculous Otolaryngeal Lesions*

	Number of Patients			
	Total	Cure	Improvement	No Change
July 1946 to December 1947				
Larynx	27	19	3	5
Mouth and pharynx	..	..	..	..
Ear	7	5	..	2
January 1948 to November 1948				
Larynx	14	12	2	..
Mouth and pharynx	1	1	..	..
Ear	4	4	..	..
December 1948 to December 1949				
Larynx	38	21	14	3
Mouth and pharynx	5	4	1	..
Ear	6	6	..	..
Totals	102	72	20	10

the severe pulmonary conditions are treated with the combined administration and intermittent therapy, more of the associated laryngeal conditions will be cleared. I have noticed no correlation between the extent of the pulmonary lesion and the healing of tuberculous otitis.

At this hospital there has been one ulcerous lesion of the nasal septum, one ulcerous lesion of the tonsil and one ulcerous lesion of the tongue, all proved by biopsy and all considered cleared with streptomycin therapy. The lesions of the tonsil healed in four weeks and that of the tongue and nose in about two months.

Wolfer and associates [36] reported one case of tuberculous ulcer of the tongue, and Shamaskin [37] reported three cases, with results of treatment similar to the case at this hospital. Lane [38] reported one case of a tuberculous granuloma

36. Wolfer, H.; Hirshleifer, I., and Shapiro, R.: Treatment of Tuberculous Ulcer of the Tongue with Streptomycin, J. A. M. A. **136**:249-250 (Jan. 24) 1948.

37. Shamaskin, A.: Tuberculous Ulcers of the Tongue: Three cases, Am. Rev. Tuberc. **56**:419-420 (Nov.) 1947.

38. Lane, C. S.: Tuberculosis of the Nose Treated with Streptomycin, Ann. Otol., Rhin. & Laryng. **58**:403-406 (June) 1949.

obstructing the left nostril. The patient was reported as having made great improvement with 1 Gm. of streptomycin administered intramuscularly daily for 23 days.

TRACHEOBRONCHIAL TUBERCULOSIS

The results of treatment of endobronchial tuberculosis with streptomycin is essentially similar to that of tuberculous laryngitis. Forsee and Erman [39] have presented the results at this hospital from June 1947 to March 1949. During this period there were 1,882 patients examined bronchoscopically, 1,108 of whom had a clinical diagnosis of pulmonary tuberculosis. Ninety-two, or 8.3 per cent, of the 1,108 patients had endobronchial tuberculosis. Further study of 24 of the 92 patients was not possible, but the condition in the remainder was classified as fibrostenotic in 19, hyperplastic in 23, submucosal in 15 and ulcerative in 11. The majority of the patients with hyperplastic, submucosal and ulcerative conditions were treated with 1 Gm. of streptomycin intramuscularly daily. The mean healing time was 42 days, 28 days for the earliest period of healing and 120 days for the longest.

EIGHTH NERVE TOXICITY

In the earlier treatment of tuberculosis with streptomycin on doses as high as 3 Gm. daily, in a high percentage of cases labyrinthine toxicity developed within a short time. The symptoms and findings were, in general, a positional type of vertigo, without spontaneous nystagmus but with bilateral depressed vestibular function and a position Romberg sign. Usually, there is no directional tendency in falling, and the optokinetic tests elicited normal responses. A small number of patients had partial loss of hearing. Little or no pathological material, either on laboratory animals or man, had been studied, so that speculation existed as to the location and extent of the pathological lesions. Considerable light has now been shed on the interpretation of the symptoms and the nature and distribution of the pathological process.

Brown and Hinshaw [40] found that vestibular dysfunction developed in about 30 days in patients treated with 3 Gm. of streptomycin daily. Deafness developed in five of their patients in from 22 to 95 days. They warned that if deafness was to be avoided the drug should be withdrawn when low-pitched tinnitus developed. Fowler and Seligman [41] found that otitic symptoms usually occurred between the seventeenth to twentieth days of treatment in patients receiving 3 Gm. daily, but they stated that the onset varied from seven to 88 days in their series. Farrington and associates [42] treated 16 patients with 3 Gm. of streptomycin daily for 120 days; in all these patients vestibular dysfunction developed, usually on about the twenty-

39. Forsee, J. H., and Erman, E. D.: Extrapleural Thoracoplasty Performed Early in Caseopneumonic Tuberculosis, Minutes of Seventh Streptomycin Conference, Denver, Colorado, April 21-24, 1949, Washington, D. C., Veterans Administration, 1949, p. 150.

40. Brown, H. A., and Hinshaw, H. C.: Toxic Reaction of Streptomycin on the Eighth Nerve Apparatus, Proc. Staff Meet., Mayo Clin. **21**:347-352 (Sept. 4) 1946.

41. Fowler, E. P., and Seligman, E.: Otic Complications of Streptomycin Therapy, J. A. M. A. **133**:87-91 (Jan. 11) 1947.

42. Farrington, R. F.; Smith, H. H.; Bunn, P. A., and McDermott, W.: Streptomycin Toxicity, Reactions to Highly Purified Drug on Long-Continued Administration to Human Subjects, J. A. M. A. **134**:678-688 (June 21) 1947.

first day of treatment. In two, deafness developed. Moffitt and Norman [43] reported on 50 patients, most of whom were treated with 2 Gm. of streptomycin daily and in whom labyrinth dysfunction developed in practically all by the thirtieth day of treatment. Another 50 patients in their series were treated with 1 Gm. daily for six to 12 weeks, and in only 4 per cent severe vestibular dysfunction developed. Peik and associates [44] found that in 21 patients treated with 2 Gm. of streptomycin daily the interval between initial loss and maximum reduction of vestibular response to caloric stimulation was one week in 90 per cent and three weeks in 100 per cent.

Thompsett [45] demonstrated in a small series of cases that vestibular dysfunction can be reduced by the use of smaller, but still effective, daily doses and also by less frequent daily divided doses. He also stated that many patients were being treated with more than the optimum therapeutic dose because the dose had not been calculated on the patient's weight.

In regard to my own findings on toxicity, I noted no depression of the labyrinth in 83 patients who finished 120 days of treatment with the combination of streptomycin and paraaminosalicylic acid on the intermittent schedule (table 1).

There has not been much human pathological material available for study, but the results of that studied together with animal experimentation lead to the conclusion that the lesions are more widespread than was at first contemplated. Mushett and Martland [46] and Glorig and Fowler,[47] in the early study of this condition, found no pathological changes present in their specimens.

Winston and associates [48] did experiments on cats that had been treated with streptomycin until they lost vestibular function. The experiments were directed toward finding any lesions of the central portion of the vestibular apparatus. Evidence of damage was found by three entirely different methods of examination:

The first method was the use of the vital staining technic. Damage was found in the Purkinje cells of the cerebellar cortex, most pronounced in the flocculus; similar findings were noted in the dentate and fastigial nuclei, the medial and lateral vestibular nuclei, the ventral cochlear nuclei, the tuberculum acusticum, the trapezoid body and the reticular substance.

The second method was that of destruction of a vestibular nucleus on one side and observation of the postoperative reaction. It had prevoiusly been shown that when a labyrinth is destroyed the ipsilateral vestibular nucleus, in a short time, compensates for this deficiency so far as tonic influence to the eye muscles are concerned and takes up this duty previously performed by the labyrinth, which is now destroyed. Thus, the authors reasoned, if the vestibular nuclei have been

43. Moffitt, O. P., Jr., and Norman, W. B.: Streptomycin Toxicity to the Labyrinth, Ann. Otol., Rhin. & Laryng. **57**:999-1006 (Nov.) 1948.

44. Peik, D. J.; High, H., Jr., and Campbell, P. A.: Streptomycin Toxicity of the Eighth Nerve, Arch. Otolaryng. **50**:251-254 (Sept.) 1949.

45. Thompsett, R.: Relation of Dosage to Streptomycin Toxicity, Ann. Otol., Rhin. & Laryng. **57**:181-188 (March) 1948.

46. Mushett, C. W., and Martland, H. S.: Pathologic Changes Resulting from the Administration of Streptomycin, Arch. Path. **42**:619-629 (Dec.) 1946.

47. Glorig, A., and Fowler, E. P., Jr.: Tests for Labyrinth Function Following Streptomycin Therapy, Ann. Otol., Rhin. & Laryng. **56**:379-394 (June) 1947.

48. Winston, J.; Lewey, F. H.; Parenteau, A.; Marden, P. A., and Cramer, F. B.: An Experimental Study of the Toxic Effects of Streptomycin on the Vestibular Apparatus of the Cat, Ann. Otol., Rhin. & Laryng. **57**:738-753 (Sept.) 1948.

rendered functionless bilaterally by streptomycin, subsequent surgical destruction of one vestibular nucleus would no longer elicit nystagmus to the opposite side as it normally would, provided that the contralateral vestibular nucleus had not already been damaged by streptomycin. The theoretical considerations were actually confirmed with streptomycin-treated cats and with adequate normal control animals.

The third method consisted of histopathological studies. Brain sections were stained with phosphotungstic acid and cresyl violet. Pyknosis and an increase in glial fibers were found around the dendrites of the Purkinje cells of the cerebellum.

Berg [49] studied the histopathological effects on the vestibular apparatus of 25 streptomycin-treated cats. The definite changes that he found localized in the sensory epithelium of the labyrinth he considered the primary lesion. The slight granular changes found also in the vestibular ganglion were considered as secondary to the more peripheral lesions.

Stevenson and associates [50] noted liquefaction necrosis and hydropic degeneration of the cells of the ventral cochlear nuclei and the inferior vestibular nuclei in streptomycin-treated dogs.

Nielson and Marsh [51] examined the brain of a patient treated with streptomycin who died of tuberculous meningitis. The ganglion cells of the dorsal and ventral cochlear nuclei and medial vestibular nuclei showed varying degrees of involvement, from chromatolysis to complete destruction. There was a minor degree of demyelination involving the cochlear and vestibular divisions of the eighth nerve, the medial longitudinal fasciculus and the medial lemniscus. The authors attributed the clinical finding of loss of association between the extraocular muscles and the neck muscles to the damage in the medial longitudinal fasciculus.

Dix and associates [52] observed 12 patients who had received prolonged treatment with streptomycin that resulted in either reduced or absent caloric responses in all. The optokinetic responses were normal in all. Because of the dissociation of caloric and optokinetic nystagmus in these cases and because of pathological reports of destruction of the vestibular nuclei, the authors conclude that one must revise the commonly accepted view that the common pathway of nystagmogenic impulses from the labyrinths and the retinas includes the vestibular nuclei. The findings of dissociated caloric and optokinetic reflexes in streptomycin-treated patients have been previously reported in three papers based on studies at this hospital.[53]

CONCLUSIONS

Treatment of otolaryngeal tuberculosis should be correlated with the treatment of the pulmonary lesions in the same patient because of the unfavorable effect

49. Berg, K.: The Toxic Effect of Streptomycin on the Eighth Cranial Nerve, Ann. Otol., Rhin. & Laryng. **58**:448-456 (June) 1949.

50. Stevenson, L. D.; Alvord, E. C., Jr., and Correll, J. W.: Degeneration and Necrosis of Neurones in Eighth Cranial Nuclei Caused by Streptomycin, Proc. Soc. Exper. Biol. & Med. **65**:86-88 (May) 1947.

51. Nielson, I. M., and Marsh, C.: Streptomycin Therapy: The Pathological Basis of Vestibular Disturbances, Bull. Los Angeles Neurol. Soc. **13**:99-103 (June) 1948.

52. Dix, M. R.; Hallpike, C. S., and Harrison, M. S.: Some Observations Upon the Otological Effects of Streptomycin Intoxication, Brain **72**:241-245 (June) 1949.

53. Moffitt, O. P., Jr.: Streptomycin Toxicity for the Labyrinth: Preliminary Report, Bull. U. S. Army M. Dept. **8**:761-765 (Oct.) 1948. Vadala.[1] Moffitt and Norman.[43]

that one lesion has on the other. The patient should not be treated with a short course of therapy aimed at clearing only the otolaryngeal lesion unless any associated lesions that require more therapy can also be treated adequately at the same time. Antibiotic and chemotherapeutic treatment of the otolaryngeal lesion should also be correlated with other necessary measures of treatment that the patient may need for associated lesions, such as rest, temporary or permanent collapse or excisional therapy.

Otolaryngeal tuberculosis often presents open lesions which are a foci for spreading tubercle bacilli to the exterior and are as much a source of public health concern as are open lesions elsewhere in the body. It appears probable that within the next few years, because of new technics—mass fluorophotographic surveys, BCG vaccination—and new methods of therapy, the time will be propitious for a new "all out" frontal attack on tuberculosis in general.

The combined therapy on intermittent and interrupted regimens holds considerable promise of reducing both toxicity and delay of emergence of resistant organisms. As newer chemotherapeutic and antibiotic drugs are investigated, it appears not unreasonable to conclude that different combinations of these tuberculostatic agents may be administered in series for as long as one year. If this supposition proves correct it is likely, then, that some of the more chronic type of lesions which have been refractory under present methods of treatment will yield under the more prolonged treatment. Otolaryngeal lesions appear to heal well in most cases with streptomycin doses between 0.5 and 1 Gm. administered intramuscularly, daily or intermittently, providing that the associated pulmonary lesion is doing well with the same treatment and unless resistant organisms emerge to vitiate the effect of the therapeutic agent.

SUMMARY

The results of treatment of 102 tuberculous patients with otolaryngeal complications are given with the various regimens used during the past 3½ years.

It appears that to date the most effective regimen for both otolaryngeal lesions and associated pulmonary lesions is one of combined administration of streptomycin and paraaminosalicylic acid on an intermittent schedule of every three days.

The above regimen appears to eliminate toxicity and to delay the emergence of resistant organisms up to 120 days of therapy.

Newer antibiotic and chemotherapeutic agents, when they have been adequately investigated, will undoubtedly extend the feasible length of treatment that can be administered in series.

The results of examination of pathological material from man and experimental animals whose labyrinthine function has been lost through streptomycin administration, as reported by different investigators, lead to the conclusion that the lesions are widespread, both in the peripheral labyrinth and in the central nervous system.

Pathological lesions have been reported to be in the sensory epithelium of the labyrinth, the vestibular ganglion, the cochlear and vestibular divisions of the eighth nerve, the cochlear and vestibular nuclei, the tuberculum acusticum, the trapezoid body, the reticular substance, the flocculus, the dentate nucleus, the fastigial nucleus, the medial longitudinal fasciculus and the medial lemniscus.

Lieutenant Colonel Kenneth Somers, MC, aided in selecting cases and in observing results.

OBSERVATIONS ABOUT THE PRESUMED ACTION OF SODIUM BICARBONATE ON THE OTOLITHIC CRYSTALS

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IN THE ARCHIVES of December 1949, Hasegawa[1] discusses his work with experimental animals and man for the prevention of motion sickness. He states that this sickness is due, above all, to abnormal stimulus of the otolithic apparatus, which expresses itself through the vagosympathetic system. To confirm this affirmation, he reminds the reader that the signs of motion sickness are lacking in animals whose otolithic crystals have been destroyed through centrifugation or in animals in which a sympathectomy has been performed. Since animals deprived of their otoliths do not show evident disturbances in balance, Hasegawa believes that destroying otoliths in man may be the best method of preventing motion sickness.

Granted that men evidently cannot undergo centrifugation, he searched for a method of practical operation that would destroy the otolithic crystals in order to make the disturbances they produce disappear.

He states that he found sodium bicarbonate, injected intravenously, to be the most suitable substance to obtain this result. To confirm this he relates the experiments carried out on rabbits and man. In the rabbits treated with 5 cc. of a 5 per cent solution of sodium bicarbonate he has observed the disappearance of motion sickness and, through histological findings, the disappearance of the otolithic crystals. After one intravenous injection of 40 cc. of a 5 per cent solution of sodium bicarbonate, some persons usually suffering from seasickness no longer felt any disturbance, even when placed in situations most conducive to seasickness. In a young man so treated, who died from sarcoma soon afterward, the histological findings showed that the otolithic crystals were absent.

With regard to the mechanism of action by which the otoliths are destroyed by the sodium bicarbonate, the author presents the hypothesis that it takes place when the carbon dioxide generated under the otolithic apparatus transforms the otolithic crystals of calcium carbonate into a soluble calcium bicarbonate. He reaches this conclusion through his personal observations that if carbon dioxide is

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1. Hasegawa, T.: Prevention of Motion Sickness by Intravenous Injection of Sodium Bicarbonate, Arch. Otolaryng. **50**:740 (Dec.) 1949.

bubbled into a test tube containing otolithic crystals in aqueous suspension, it is not possible to show their presence by microscopic examination.

Given the remarkable theoretical and practical interest of the subject, we wanted to assure ourselves that the sodium bicarbonate really has an action on the otolithic apparatus. Hasegawa's histological observations were, a priori, scarcely reliable because they were obtained from decalcified and then sectioned labyrinths; the absence of otolithic crystals (constituted of calcium carbonate) could be due to their dissolution, owing to the substance used for the labyrinth's decalcification.

Having ascertained in vitro that otolithic crystals placed in a 5 and 9 per cent solution of sodium bicarbonate (the 9 per cent being the maximum concentration at room temperature) could be observed microscopically, even after 24 and 48 hours, and having, on the other hand, uncertain results with the test of the bubbling of the carbon dioxide, according to Hasegawa's technic we treated a series of pigeons with intravenous injections. In these animals the otolithic apparatus is very rich in otolithic crystals and the posterior labyrinth is remarkably well developed, anatomically and physiologically. It was easy to penetrate their vestibulum, to cut off their otolithic membranes and to carry out histological examinations *a fresco* and without decalcification. In this way the eventual disappearance of the otoliths could be logically attributed only to the dissolving action of the sodium bicarbonate. Moreover, we also treated a series of guinea pigs, which have, as is well known, good otolithic reflexes. The treatment consists in injecting into the animals' vein various quantities of sodium bicarbonate solution at different concentrations and in observing histologically, as with pigeons, whether the otolithic crystals were really destroyed. With the guinea pigs we looked for the disappearance of the otolithic reflexes, which would demonstrate an action of the sodium bicarbonate on the otolithic apparatus.

Following are the results of our researches.

Into the vein of the wing of a first series of five pigeons were injected various (from 2 to 10 cc.) doses of a 5 per cent solution of sodium bicarbonate, as proposed by Hasegawa. The animals were killed 24 to 72 hours after the injection, according to a schedule shown in the following tabulation:

Pigeon	5 Per Cent Solution	Period Before Being Killed
1	2 cc.	24 hr.
2	2 cc.	72 hr.
3	5 cc.	24 hr.
4	5 cc.	48 hr
5	10 cc.	72 hr.

As soon as the animals were killed, the labyrinths were cut open and the otolithic membranes removed and immediately observed with the microscope. In every case we always observed that the otolithic crystals were still present and had the same morphological characters as those of the normal control pigeons.

We then treated a second series with the more concentrated solution of 9 per cent:

Pigeon	9 Per Cent Solution	Period Before Being Killed
1	3 cc.	48 hr.
2	5 cc.	60 hr.
3	5 cc.	72 hr.

The injection of 5 cc. of the 9 per cent solution was immediately followed by the same symptom complex as in the alkalotic coma, so we did not think it necessary to use higher doses. Histologically, the results in this series were the same as in the preceding one.

We then treated the third series of pigeons with repeated injections of sodium bicarbonate solution:

	First Injection		Second Injection		Third Injection		
Pigeon	Strength Solution	Amt., Cc.	Strength Solution	Amt., Cc.	Strength Solution	Amt., Cc.	Period Before Being Killed
1.....	5	2	5 (After 24 hr.)	10	..	..	72 hr.
2.....	5	5	5 (After 48 hr.)	5	5	5	48 hr.
3.....	5	5	9 (After 72 hr.)	5	..	..	72 hr.
4.....	5	10	9 (After 48 hr.)	5	..	..	72 hr.

The histological examinations carried out in this series with the same technic used previously showed that the otolithic crystals still existed.

In all instances the histological tests were carried out, as can be noted in the above tabulations, no longer than 72 hours after the last treatment, since we observed, through p_H measurements of the blood, that the alkalosis lasted no longer than 48 hours.

Therefore, we thought that any action of the sodium bicarbonate could be considered completely exhausted in 72 hours.

Figure 3 shows the variations of the blood p_H observed in relation to the doses and to the concentration of the solutions used.

From the results it was evident that the otolithic crystals are not destroyed by sodium bicarbonate, even if the solution is injected repeatedly in doses and concentrations higher than those suggested by Hasegawa. We sought to determine whether the sodium bicarbonate, though unable to dissolve the otolithic crystals, had any action on their function by checking the reflexes originated by them. In such a case an action against seasickness could be explained.

Because of the accessibility of the vestibular reflexes of the guinea pig for investigation, we chose it as the experimental animal.

We treated a series of guinea pigs by injecting into the jugular veins various doses of sodium bicarbonate solution in various concentrations and tested the otolithic reflexes before the treatment in order to determine the normalcy of their function. Immediately after the treatment and 24, 48 and 72 hours after the treatment, we repeated the examinations and compared the results with those obtained in both a guinea pig with a destroyed labyrinth and in a control guinea pig. We kept under control the compensatory eye positions, righting reflexes, tonic labyrinthine reflexes and reactions to vertical acceleration.

In every animal treated with solutions of sodium bicarbonate in doses varying from 5 to 10 cc. and concentrations from 5 to 9 per cent, the reflexes examined proved to be perfectly normal, as were the reflexes of a guinea pig treated with 10 cc. of a 9 per cent solution, although this guinea pig immediately showed symptoms of alkalotic coma.

Figure 4, of a normal untreated guinea pig (*A*), of the treated one (*B*) and of one with a destroyed labyrinth (*C*), shows the righting reflex.

Fig. 1.—Otolithic crystals of untreated pigeon.

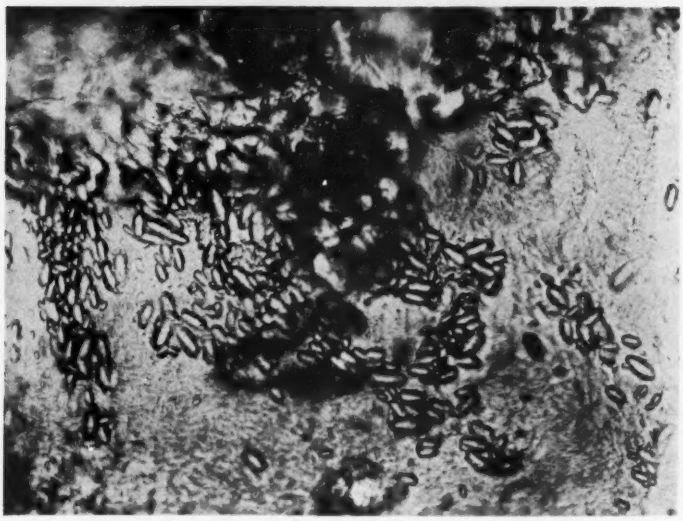

Fig. 2.—Otolithic crystals of a pigeon in which injections were made of 5 cc. of a 9 per cent solution of sodium bicarbonate.

In the most evident way it has been shown how the treated guinea pig behaves exactly like the normal one and very differently from one without a labyrinth.

Thus the experiments show that the otolithic reflexes are neither inhibited nor modified by sodium bicarbonate.

Therefore, on the basis of these results, the possibility that sodium bicarbonate has any action on the otoliths and otolithic reflexes can be excluded.

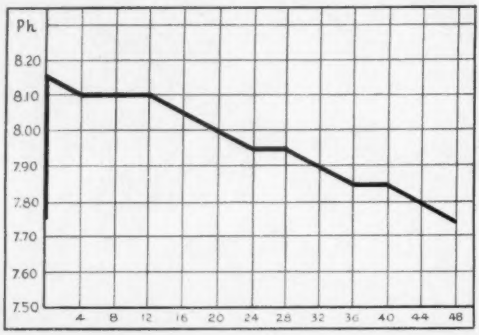

Fig. 3.—Variations of the blood p_H observed in relation to the doses and to the concentration of the solution used.

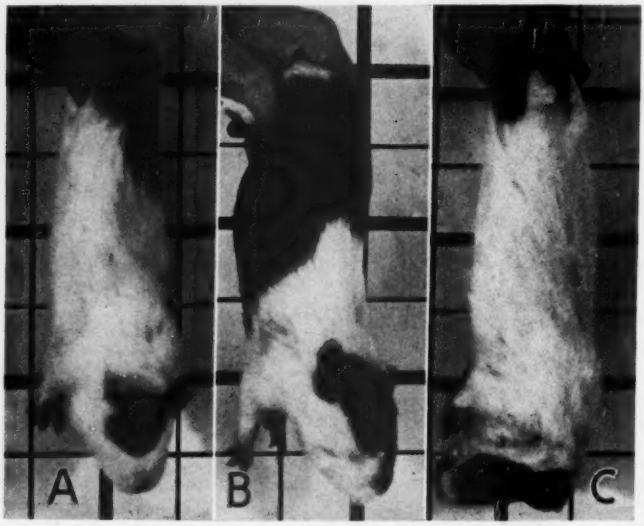

Fig. 4.—*A*, normal untreated guinea pig; *B*, guinea pig treated with sodium bicarbonate, and *C*, guinea pig with a destroyed labyrinth.

SUMMARY

Investigations carried out on pigeons and guinea pigs show that, in spite of Hasegawa's results, sodium bicarbonate injected intravenously has no direct action on the otolithic crystals and does not affect their function, even when solutions in remarkably high doses and concentrations are used.

INCIDENCE AND CONTROL OF TRAUMA ACCOMPANYING ENDOTRACHEAL ANESTHESIA

A Survey Based on the Response of One Hundred and Fifty Leading Laryngologists and My Experience of Twenty-Five Years in the Use of the Method

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THE FIRST report of my experience in the use of endotracheal inhalation anesthesia appeared in 1927.[1] The instrumentarium reported in 1928 [2] has been in constant use since that time. In 1937 [3] a report based on 10 years' use of this method was accorded 25 pages of text. The present report, covering an experience of 25 years, not only confirms the experience of the first 10 years but with all humility offers this experience to those who have suffered regrets in the use of other technics and instruments.

A letter received from Dr. Chevalier Jackson dated Dec. 17, 1926 struck a prophetic note: "The great trouble has been that instead of taking a few lessons on exposure of the larynx with the laryngoscope, nearly everyone who has tried intratracheal anesthesia starts out with devising some new tube or some new method of introduction." Viewing the morbidity and the mortality described in the report which follows, one might add that there is an increasing tendency to subordinate the anatomy and the physiology of the laryngeal structures to the mechanical needs of new anesthetic agents and anesthetic gadgets.

The use of endotracheal tubes and the technic of intubation begun under the supervision of Dr. Jackson in 1926, based as these were on the anatomic capacity and the physiological tolerance of the glottis, have demonstrated in my hands their advantages and left me with a unique freedom from regrets.

During the last quarter century, endotracheal anesthesia for surgery of the head and neck and for purposes of resuscitation has become mandatory. It will not be surprising if, within the near future, a failure to employ these procedures will be construed as malpractice.

Because of this impending universal use, something must be done to control the appalling mortality and morbidity which the present investigation lays bare. In the spring of 1950 a questionnaire was sent to 514 members of the American

1. Flagg, P. J.: Intratracheal Inhalation: Preliminary Report of Simplified Method of Intratracheal Anesthesia Developed Under Supervision of Dr. Chevalier Jackson, Arch. Otolaryng. **5**:394-399 (May) 1927.

2. Flagg, R. J.: Intratracheal Inhalation Anesthesia: Method of Election for Operations on Head and Neck, Arch. Otolaryng. **7**:156-164 (Feb.) 1928.

3. Flagg, P. J.: Intratracheal Inhalation Anesthesia: Review of 10 Years' Experience, with Special Reference to Its Field of Usefulness, Details of Technic and Objections Raised Against Method, Arch. Otolaryng. **25**:405-429 (April) 1937.

Laryngological, Rhinological and Otological Society. The responses suggest that disaster is invited by the use of oversized tubes, by the use of endotracheal instead of pharyngeal packing and by blind intubation. It is believed that a correction of these errors will result in a major elimination of the pathological conditions reported.

It has been repeatedly pointed out that the introduction of a new drug (anesthetic) involves many factors of a biochemical, physiological and histological nature. Safety, the therapeutic index of the new drug, is arrived at only after a long period of use and abuse; witness tribromoethanol (avertin®) and numerous intravenous anesthetics. On the other hand, the safety of a mechanical procedure, the success of which turns on physical factors and the local reaction of the tissues, is much more quickly and accurately established.

In the first instance an intimate knowledge and an acceptance of the experience of pharmacology and therapeutics is implied and must parallel the surgical and anesthetic skill of the laryngologist. In the second, the procedure turns on the technical skill of the operator. To determine precisely the anticipated reaction in the employment of premedication, intravenous administration of thiopental (pentothal®) sodium, curare and local administration of cocaine, is a much more complicated matter and presents a much narrower margin of safety than does the mechanical intubation of the larynx. The first remains within the experience of pharmacology and therapeutics, regardless of hit or miss surgical trials. The second, established by accepted anatomic and physiological standards, is perfected by natural or acquired technical skills. It is therefore believed that, in contradistinction to the difficulty of eliminating the hazards of drug anesthesia, the problem of the hazards of endotracheal intubation is readily solved. An experience of 25 years in my hands, as well as in students' hands, suggests that the skill necessary to examine with a laryngoscope and intubate the unconscious relaxed patient does not exceed that required for the inspection and the treatment of any other closed cavity, i.e., ear nose, bladder, rectum, etc. The orientation of the field, respect for the tissues involved, restraint and temporary postponement of intubation when conditions are not suitable may easily be acquired by any one with average technical dexterity.

Brief relaxation with short acting anesthetics complicated by impending or existing respiratory failure from massive administration of drugs for sedation and curare in shock doses creates pressure reducing the available time for intubation. Under these conditions intubation becomes much more than a technical procedure. It is the only means of meeting an acute asphyxial emergency. While this communication does not admit of a detailed consideration of preintubation anesthesia, my experience has been that the barbiturates increase the laryngeal reflexes and the addition of the curare necessary to suppress these reflexes induces severe respiratory depression with its stagnant anoxia. I have used routinely gas-ether or tribromoethanol; for ether induction, an ether maintenance supplemented with intravenous administration of morphine sulfate, scopolamine and ephedrine. I have felt that the convenience and speed of a cyclopropane or pentothal-curare induction with premedication does not compensate for the immediate and the late hazards involved. I am well aware that these views are not shared by many surgeons and anesthetists who may feel justified in the conclusion that perhaps, after all,

an occasional death or laryngeal injury may not be too high a price to pay for the great number of satisfied patients who escape.

THE INCIDENCE OF TRAUMA FOLLOWING ENDOTRACHEAL INTUBATION

Assuming that the laryngologist is familiar with laryngeal anatomy, physiology, tolerance to trauma and postoperative reaction to instrumentation and that this familiarity is more intimate than is that of other members of the medical profession, anesthetists included, a questionnaire was mailed to every member of the American Laryngological, Rhinological and Otological Society, a total of 514. A response was received from 154 laryngologists. This 30 per cent response actually reflects a much larger percentage of the total group of laryngologists, because many of the members of the society are engaged in rhinology and otology rather than laryngology. Because bronchoscopy is ordinarily performed in the conscious patient against muscular resistance and frequently against more or less reflex spasm, because the bronchoscopic tube is much longer, completely rigid and in the course of its use rubs constantly against the endotracheal mucosa without benefit of lubricant, it would be fair to assume that the postoperative trauma to be expected would exceed that following the use of the short, flexible, lubricated tube used for endotracheal anesthesia. Furthermore, in spite of the longer period of intubation, the tube, once placed, is comparatively stationary. In spite of this assumption, the responses to the question, What laryngeal and tracheal reaction do you expect in the course of ordinary laryngoscopy? were no reaction, 80; slight reaction, 53 and severe reaction, 2.

In summary, little or no reaction is expected by 98 per cent of the respondent laryngologists. An exception is that much more reaction is expected in infants and very small children.

The questionnaire then poses the following question, Have you been called in consultation for an opinion or for the treatment of a pathological condition of the larynx or trachea following endotracheal intubation for anesthesia, i. e., granulomas of the cords, lacerations of the cords, injury to mucosa or carina of the trachea or other forms of injury? The answers to this broad inquiry are tabulated as follows: number of surgeons called in consultation, 99; number not called, 39; no reply, 14. Thirty-four surgeons reported a total of at least 101 granulomas. (A number replied yes without stating the number of granulomas.) The following special injuries were reported by 31 men: hemorrhage of vocal cords (2 cases), irritation from tubes soaked in benzalkonium (zephiran®), injury to mucosa of trachea (3 cases), lacerations of cords (5 cases), edema of cords (5 cases), hoarseness, mucosa scraped from cords, trauma to carina trachea, edema of trachea (2 cases), paralysis of cords, perforation of trachea (2 cases), laceration of cord, laryngeal edema, tracheal webs (3 cases), leukocytic membrane of cord and trachea and traumatic laryngitis.

To the question, Have you encountered any of the above lesions in routine bronchoscopy? the following replies were received: no, 107; yes, 25; no reply, 21. Of the total replying (132), 80 per cent did not meet these lesions in routine bronchoscopy. (The lesion was commoner in the infant and small child group.)

To the question, Have these lesions appeared immediately or some time after intubation? the answers were edema, after two to 12 hours; acute lesions, immediately; granulomas, after four weeks to five months.

The problem of emergency relief by tracheotomy was raised in the following question, Have you been obliged to do a tracheotomy for trauma? Forty-two respondents, approximately 30 per cent, were obliged to do tracheotomies. Because a consultant is not the operating surgeon or necessarily present at an accident, the following question was asked, Are you aware of deaths resulting from endotracheal anesthesia? Of the 132 who answered this question, 24 answered yes and 10 considered intubation a factor. Because of the widespread location of the physicians who replied, it is not likely that there was much, if any, overlapping in the case sources. In view of the responsibility of the respondents, most of these men being chiefs of services, a "yes," while the meaning is open to discussion, suggests a situation worthy of special investigation.

The replies to two more questions remain to be considered. The first deals with technic, the second with instruments. The question was posed, Do you favor blind (intranasal) intubation? The answers were 111, no; 8, yes; 16, yes and no; 16, no response. Of those who had definite opinions, 90 per cent replied no; 10 per cent replied yes. Fourteen of the no's were emphatic, i. e., absolutely not: "I have often suspected that some damage has been done to the cords by blind intubation"; "No"; "Definitely no"; "Definitely no; last week a patient complained bitterly of intranasal intubation"; "In one instance a false passage was made by the tube perforating the mucous membrane above Passavant's band, with the production of a false passage down to the hypopharynx"; "My associates and I had one case in which atelectasis developed after this procedure; we are inclined to believe this was due to a large crust that had formed in the nasal cavity and was forced down into the trachea by blind intubation"; "Definitely opposed to blind intubation."

The last question is believed to be most important. This question solicits the judgment of the laryngologist concerning the maximum size (outside diameter) of the endotracheal tube to be used in infants, 2 year old children, 5 year old children, 10 year old children and adults. Because of the importance attached to these opinions, the responses are reported in detail.

Infants—2.5 mm. (5 laryngologists), 3 mm. (27), 3.5 mm. (15), 4 mm. (8), 6 to 7 mm. (1)

Two year old children—3.5 mm. (8 laryngologists), 4 mm. (24), 5 mm. (13), 6 mm. (4), 7 to 8 mm. (2), 8 to 9 mm. (2)

Five year old children—3 mm. (6) laryngologists), 4 mm. (11), 5 mm. (30), 6 mm. (10), 7 mm. (4), 8 to 9 mm. (3)

Ten year old children—4 mm. (2 laryngologists), 5 mm. (12), 6 mm. (19), 7 mm. (13), 8 mm. (5), 9 mm. (2), 10 mm. (4)

Adults—5 mm. (2 laryngologists), 6 mm. (1), 7 mm. (18), 8 mm. (16), 9 mm. (17), 10 mm. (9), larger than 10 mm. outside diameter—12 mm. (3) (one of these was the anesthetist who reported for the surgeon), 13 to 15 mm. (3)

The data can be summarized in the following way:

Infants—55 laryngologists (95%) limited tube O.D.[4] to 4 mm.
Two year old children—51 laryngologists (84%) limited tube O.D to 5 mm.
Five year old children—57 laryngologists (89%) limited tube O.D. to 6 mm.
Ten year old children—48 laryngologists (89%) limited tube O.D. to 8 mm.
Adults—65 laryngologists (92%) limited tube O.D. to 10 mm.

Over-All Summary of Questionnaire.—Trauma Following Bronchoscopy: In routine laryngoscopy and intubation for bronchoscopy 98 per cent of the respondents expected little or no reaction.

4. Outside diameter.

Consultations for Trauma Following Anesthesia: Ninety-nine respondents, or 64 per cent, were called in for postoperative trauma following endotracheal intubation for anesthesia.

Pathological Conditions Following Intubation for Anesthesia: Thirty-four surgeons reported a total of 101 granulomas following intubation for anesthesia. Thirty-one reported a variety of lesions.

Pathological Conditions Following Bronchoscopy: Eighty per cent of the respondents have not encountered the above lesions in routine bronchoscopy. Twenty per cent encountered minor lesions (except in infants).

Time of Appearance of Lesions: Acute lesions appeared immediately or from two to twelve hours after intubation; chronic lesions and granulomas appeared from four weeks to five months later.

Necessity for Tracheotomy: Thirty per cent of the respondents were obliged to do tracheotomies for trauma.

Deaths: Twenty-four respondents replied that they were aware of deaths having followed intubation for anesthesia. Ten replies suggested that fatalities were due to intubation.

Blind Intubation: Ninety per cent condemned blind intubation. The maximum outside diameters in endotracheal tubes were recommended. Ninety-five per cent restricted the infant's tube to 4 mm. outside diameter. Ninety-two per cent limited the adult's tube to 10 mm. outside diameter.

COMMENT

The over-all absence of trauma from routine bronchoscopy and the relatively high mortality and morbidity in endotracheal anesthesia reflected in this report suggest an immediate search for the cause. The vigorous condemnation of blind (intranasal) intubation, 90 per cent of our respondents being opposed, is worthy of serious consideration. The responses regarding the maximum outside tube diameter (not bore or inside diameter) are considered fundamentally important. If 95 per cent of the respondents restrict the infant's tube to 4 mm. outside diameter and 92 per cent restrict the adult's tube to 10 mm. outside diameter, how can the use of endotracheal tubes for anesthesia which exceed these measurements be justified?

The following makes and sizes of tubes are in routine use.

Magill—red rubber and portex 3.75, 4.5 to 13 mm.
Latex, spiral 7 mm. to 13 mm.
Latex, spiral; cuff built in 7 mm. to 15 mm.
Latex, spiral; with balloon (for adults) to 17 mm. O.D.[4]
Silk, woven 4.33 to 13 mm.

It is customary to measure endotracheal tubes for anesthesia by internal diameter, or bore. In other words, the ventilation through the tube rather than its size as a foreign body has been stressed.

In addition to the trauma produced by blind intubation and by the common use of oversized endotracheal tubes, a third factor is undoubtedly responsible for much of the trauma. It lies at the basis of the causes and is largely responsible for the use of the oversized tubes. This third factor becomes clear in the following consideration.

Endotracheal intubation for anesthesia has become indispensable for two reasons: it insures suitable ventilation and guards against aspiration of fluids, especially in operations on the head and neck. Ventilation requires intubation. A 7 to 9 mm. internal diameter tube is fully adequate. Tubes are available with a rigid wall of only 0.5 mm. thickness. The recommendations of the laryngoscopists of limiting tubes to 10 mm. outside diameter are not unreasonable.

Aspiration around the endotracheal tube, which but partly fills the glottic chink, may be prevented in two ways: (1) by the use of an oversized tube and by the use of an endotracheal subglottic pack or balloon distended after intubation beyond the cords and (2) by applying a gauze pack around the tube in the hypopharynx. Since oversized tubes and/or balloon packs cause trauma, the use of the pharyngeal pack is indicated. The pack is easily applied. When moistened it does not cause trauma. It insures ventilation through the endotracheal tube. It prevents the regurgitation of gastric contents from below and the seepage of saliva and blood from above from entering the trachea. (When bronchial segregation by endobronchial balloon is indicated, the pack can be used in addition as protection against leakage into the trachea. Fluid which may escape from the pack into the trachea is readily removed by endotracheal suction.)

My technic and instruments have been fully described.[5]

On the initial recommendation of Dr. Chevalier Jackson and by reason of previous experience with intrapharyngeal insufflation and inhalation, I have not used blind intubation. Intranasal intubation by direct vision is practiced in difficult exposures due to trismus or jaw injuries. The endotracheal tube employed has been a modification of the standard bronchoscope. The outside diameter has never exceeded 10 mm. (Only 1 mm., total, is sacrificed to the walls of these tubes, that is, a tube with a 10 mm. outside diameter has a 9 mm. bore and one with a 4 mm. outside diameter has a 3 mm. bore.) Endotracheal packs by balloons have not been used. Pharyngeal packing has been routine procedure. Dry gauze packed into the pharynx is hygroscopic and irritating. The use of wet gauze leaves no regrets.

In my experience intubation has paralleled routine bronchoscopy in its freedom from reaction, immediate or late. The small outside diameter of the metal tube is well tolerated by infants and little children, who as a group have presented no difficulties when well relaxed by ether anesthetic. A side light on my conviction that the technic which I employ does not cause trauma will be found in my long reported experience in dental cases of election.[5] In these cases certainly postoperative lesions could not be tolerated or condoned. A year's dental work has often been done in one four-hour period without regrets.

CONCLUSIONS

For the last 25 years I have been profoundly grateful for the safety and control afforded by endotracheal inhalation anesthesia. Because of the freedom from complications, both early and late which I have enjoyed, I have found no reason to elaborate or to change my technic or instruments. Unable to reconcile accidents

5. Flagg, P. J.: A Year's Dental Work in One Morning While the Patient Sleeps, New York J. Dent. **12**:198 (May) 1942; Safe General Anesthesia for the Patient Who Must Have Year's Dental Work Done in One Afternoon, Ann. Dent. **6**:214-219 (Dec.) 1947.

and injuries currently reported with my experience, fearing for the good name of a priceless safety factor in surgery and fully aware that there was no mystery or special skill in my use of the method, I have attempted to offer an explanation for the good fortune I have enjoyed. It would seem that endotracheal intubation for anesthesia should cause less trauma than bronchoscopy. The responses to the questionnaire prove the contrary. What, then, are the factors which have entered into intubation for anesthesia but which are not present in bronchoscopy? These factors are three: blind intubation, oversized tubes and endotracheal packing. Since these three factors are recognized as potential hazards and as they are not essential to good endotracheal anesthesia, perhaps their elimination in whole or in part may help to reduce the regrets reported and serve to protect the value of a life-saving technic.

I am fully aware that this paper is but a straw in the wind and that to offer my personal experience in support of a policy is much like the only man in the regiment who was in step; nevertheless the laryngologists who have cooperated in this survey do deserve a respectful and thoughtful audience. After all, these men represent 150 opinions, I but one.

205 East Seventy-Eighth Street (21).

Case Reports

SCLEROMA OF THE NOSE AND LIP

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NEWARK, N. J.

RHINOSCLEROMA is a benign chronic proliferating granuloma which has its origin in the mucous membrane of the upper respiratory tract. It was first described by von Hebra [1] and the causative organism described by von Frisch.[2]

A perusal of the literature revealed the disease to be a rarity in people born and raised in the United States but rather a common entity in eastern Europeans and South and Central Americans. Reyes [3] in El Salvador, Central America, has reported his observations of 200 personal cases, 120 of which were in women. He states that, "Rhinoscleroma is a disease contracted through poor personal hygiene; it is never found among people of absolutely clean habits. Its contagiousness is doubtful."

Thorough reviews of the literature with excellent bibliographies and summaries of cases in the United States were reported by Gadomski,[4] Weiss [5] and Cunning and Guerry.[6] The latter reviewed the cases of 102 patients, of whom only 16 patients were native-born Americans. Hara, Pratt, Levine and Hoyt [7] reported seven cases of scleroma in their different stages of development in members of a single family. Goldstein [8] and Gadomski [4] also reported single cases of scleroma in patients residing in New Jersey.

After reviewing the literature it is apparent that more cases of rhinoscleroma are being reported. Either the otolaryngologist is better able to recognize the disease or the disease is becoming more prevalent. It is the purpose of this article to report another case of scleroma.

Reyes [3] described the course of rhinoscleroma as occurring in three stages: first stage, the rhinitis period; second stage, the infiltrating period, and third stage, the nodular period. These three periods take many years to reach full development.

REPORT OF A CASE

Mrs. A. B., a Russian born Jewess aged 52, was first examined at my office on April 28, 1947. She was referred by her attorney for a medicolegal examination of her nose and upper lip. The patient stated that on Sept. 25, 1945 she was struck by a taxicab, knocked unconscious

1. von Hebra, F.: Rhinosclerom, Wien. med. Presse **11**:445, 1870.
2. von Frisch, A.: Zur Aetiologie des Rhinoskleroms, Wien. med. Wchnschr. **32**:969, 1882.
3. Reyes, E.: Rhinoscleroma: Observations Based on a Study of 200 Cases, Arch. Dermat. & Syph. **54**:531 (Nov.) 1946.
4. Gadomski, C. F.: Rhinoscleroma: Report of a Case, Arch. Otolaryng. **29**:681 (April) 1939.
5. Weiss, J. A: Scleroma (Rhinoscleroma): Histologic Changes Following Teleradium Therapy: Review of Scleroma in the United States, Arch. Otolaryng. **30**:38 (July) 1939.
6. Cunning, D. S., and Guerry, D., III: Scleroma, Arch. Otolaryng. **36**:662 (Nov.) 1942.
7. Hara, H. J.; Pratt, O. B.; Levine, M. G., and Hoyt, R. E.: Scleroma: A Clinico-Pathological Study of Seven Cases in One Family, Ann. Otol., Rhin. & Laryng. **56**:769, 1947.
8. Goldstein, H. Z.: Rhinoscleroma in New Jersey with a Case Report, J. M. Soc. New Jersey **38**:581, 1941.

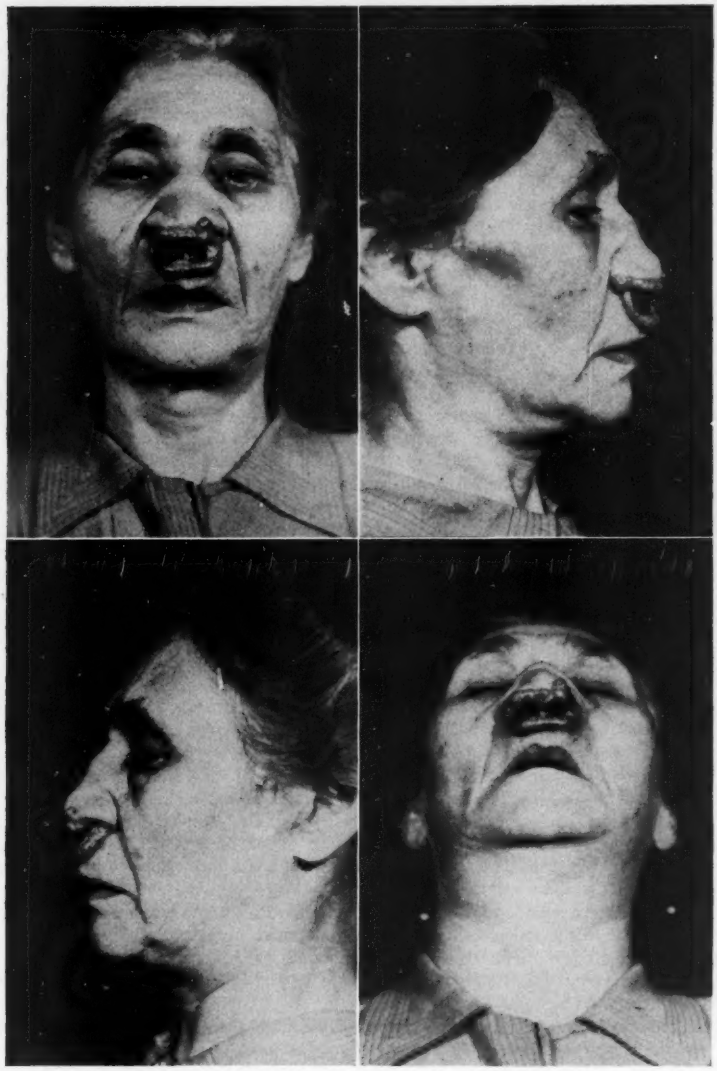

Fig. 1.—Note the typical post-traumatic saddle deformity of the nose. Both nares were completely occluded by the granulomatous mass which involved the columella, both alae and the greater portion of the lip. Pea-shaped nodules seemed to have fused to form the upper rim of the lesion. The lower border is firm, annular and keloidal in character.

and taken to St. Michael's Hospital in Newark. Her nose was smashed inwardly, and her upper lip was lacerated. Her left arm was so badly mutilated that Dr. Samuel Ebenfeld was forced to amputate it. The patient recovered and was discharged from the hospital on October 30. She further stated that in May 1946 her nose started to swell, and nasal breathing became impossible. "Loose flesh" proliferated from her nose and grew until she was unable to get any air into her nose. No physician was consulted, and no treatments were given (fig. 1).

I made the following medicolegal report on April 28, 1947. "The nose is elongate and broad with a dorsal hump and a depressed saddle fracture of the middle one-third of the dorsum of the nose. Both nares are completely occluded by a granulomatous mass. This mass involves the columella, rim of both alae and the tip of the nose. It extends downward over the upper five-sixths of the upper lip. The growth on the lip measures 4½ cm. x 2 cm. It is purplish in color and annular in outline. Small pea-shaped nodules have apparently fused to form the upper rim of the lesion. A few discrete nodules are present along the alae. The lesion is neither tender nor painful to touch. The lower border is smooth, firm, elevated and keloidal in character. The center of the lesion presents a raw granulating surface covered by a brownish sanguinous exudate. No lymphadenopathy is present. No biopsy or other tests were permitted. The nasopharynx, oropharynx and larynx were normal. The tentative diagnosis is:

1—Post-traumatic deformity of the nose and nasal bones

2—Infected granuloma of the nose and upper lip of undetermined etiology."

On November 3, the patient reappeared at my office for diagnosis and treatment. She complained of inability to breathe through her nose, recurrent mild postnasal hemorrhages and an odoriferous bloody discharge from the lesion on her upper lip. She was compelled to wear a bandage continuously on her upper lip. The patient was questioned more thoroughly at this time and admitted having two nasal operations at the Newark Eye and Ear Infirmary.

A report was received from the infirmary that on Jan. 1, 1930 the patient was admitted to the hospital for submucous resection. She had a nasal obstruction, the turbinate bones were hypertrophied and the septum was thin. The microscopic report stated that the section studied was composed of fibrous and fibroblastic tissue stroma in which there were numerous normal or slightly hypertrophic gland structures which were infiltrated by pus cells. The epithelium covering the tumor showed slight ulceration. There was an acute inflammatory nasal polyp. January 19 she was discharged from the hospital. Aug. 18, 1938 she was readmitted to the hospital. Granulations were present in the right side of the nose occluding the breathing space, and the septum appeared to be pushed over to the left. A biopsy was taken and removal of granulations inside the nose was done. A diagnosis of chronic granuloma of both nares, type unknown, was made. August 20 she was discharged from the hospital. September 15 a culture of material from both nares did not yield Corynebacterium diphtheriae. The reactions in the Kahn test were negative.

After receiving this report, I suspected that I was dealing with a slow growing granuloma which was present 17 years previously. Several biopsies of the lip lesion were taken, and Dr. William Antopol, pathologist of the Newark Beth Israel Hospital reported that it was a granuloma with macrophages containing a higher order of bacteria resembling fungus. Identification should be made on cultural characteristics of a specimen examined by bacteriologic methods. He advised culture on Sabouraud's medium.

Six cultures grown from smears of the granuloma were reported negative. Finally cultures made from pieces of the lesion itself revealed the causative organism. Philip Beerman, bacteriologist of the Newark Beth Israel Hospital reported that it was Klebsiella rhinoscleroma, and that the identity of this organism was confirmed by Dr. D. T. Smith, mycologist at Duke Hospital, Durham, N. C.

Sinus and chest roentgenograms showed no abnormalities. Blood examinations, Wassermann tests and urinalyses revealed no abnormalities except a trace of sugar in the urine. Inspection of the nose revealed the lesion to have proliferated since the last examination. It had spread locally to involve the vermilion border of the upper lip. It had also spread posteriorly to occupy a part of the nasopharynx. The uvula, oropharynx, hypopharynx and larynx were not involved.

The possibility of excising the nasal masses and reconstructing the nasal cavities by skin grafts was considered. However, it was deemed advisable first to explore the interior of the nose to determine whether the growths were limited to the anterior and posterior choanae or

extended continuously throughout the nasal passages. Under block and infiltration local anesthesia a tract was electrocoagulated through the anterior mass. It was continued posteriorly for approximately 2 inches (5 cm.). The septum and turbinate bones were indistinguishably matted into the granuloma. No nasal anatomic structures could be defined. It seemed futile to continue, and this procedure was abandoned. However, since the lip lesion had so rapidly proliferated to involve the vermilion, it was thought advisable to attempt to destroy it locally. Under bilateral block anesthesia the whole lip lesion was thoroughly destroyed by electrocoagulation. Potassium iodide, 5 grains (0.32 Gm.) three times a day, was administered. The lip lesion healed rapidly and satisfactorily, with considerable cicatrization (fig. 2). The patient has been observed for two years after this procedure. There no longer appears to be any tendency toward proliferation, and the disease appears to be dormant. The postnasal hemorrhages have ceased. The patient feels better and is more comfortable, since it is no longer necessary to wear a lip bandage.

Fig. 2.—Two years after destruction of part of the lesion by means of electrocoagulation. Note the cicatricial protrusion of the upper lip.

Devine, Weed, Nichols and New [9] recently reported a good result after using streptomycin as the therapeutic agent in a patient who had an early stage of rhinoscleroma.

COMMENT

The external appearance of the nose of this patient is typical of a post-traumatic saddle deformity. However, the broadened depressed lower cartilaginous half of the nose has been described by other observers as part of the later stages of the

9. Devine, K. D.; Weed, L. A.; Nichols, R. N., and New, G. B.: Rhinoscleroma Apparently Cured with Streptomycin, Proc. Staff Meet., Mayo Clin, **22**:597, 1947.

disease. In this case it is impossible to determine whether the saddle deformity is post-traumatic or the result of destruction and cicatricial saddling due to the scleromatous processes.

Since it is difficult to isolate von Frisch's organism, it is probable that the disease is commoner than reported. In this case the disease probably proliferated from the nose to the upper lip under the stimulus of trauma and the lacerations of the lip.

SUMMARY

A patient with scleroma has been reported. This is the third case to be reported in the state of New Jersey.

After electrocoagulation of a portion of the lesion and the administration of potassium iodide, the disease seems to have become dormant.

31 Lincoln Park.

CEREBROSPINAL RHINORRHEA FROM CRANIOMASTOID FISTULA

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WILLIAM D. FARMER, M.D.
AND
WARDELL H. MILLS, M.D.
GREENSBORO, N. C.

LEAKAGE of cerebrospinal fluid from the nose is by no means a rare sequel of head trauma. Such a finding is generally regarded as pathognomonic of a communication in the region of the paranasal sinuses or cribriform plate. It is emphasized, with justification, that failure to visualize a fracture line on the roentgenogram does not disprove such a diagnosis.

It has been shown, however, that in rare instances rhinorrhea may result from a communication between the subarachnoid space and the mastoid or middle ear, the fluid passing through the eustachian tube and into the nasopharynx.[1] The following case report illustrates this situation and, we believe, offers a means by which it can be recognized.

REPORT OF A CASE

A girl, aged 6, sustained multiple injuries when struck by a car on Nov. 10, 1949. Examination three days following the injury revealed ecchymosis over the right mastoid, paralysis of the right side of the face, and of the right lateral rectus muscle. Otoscopic examination showed the ears to be apparently normal; there was hearing loss in the right ear. With the head lowered, clear colorless fluid drained from the left nostril at a rate of 30 to 40 drops a minute. This fluid was found to contain sugar, establishing its identity as cerebrospinal fluid.

Skull films showed a linear fracture of the right occipital bone extending into the body of the petrous portion of the temporal bone. The right mastoid antrum was cloudy. Views of the paranasal sinuses and anterior fossa failed to demonstrate a fracture.

Prophylactic therapy with the use of sulfadiazine and penicillin was started. Early surgery was not considered because of associated injuries (fractured humerus, multiple rib fractures and contusion of the lung). During the subsequent two weeks, the child's general condition improved. Rhinorrhea persisted, with drainage noted from the right nostril as well as the left. The cranial nerve signs remained unchanged. At no time did signs of meningitis develop.

It was felt that surgery was necessary in view of the continuing rhinorrhea. The available evidence pointed to the posterior fossa as the site of the lesion, but incontestable proof was lacking. Routine examination of the nose was not helpful, but it was suggested by one of us (W. D. F.) that catheterization of the eustachian tube might be of value. Accordingly, this procedure was planned as a preliminary to operation.

Operation (Nov. 26, 1949).—With the patient under endotracheal ether anesthesia, catheterization of the right eustachian tube was carried out with the head lowered. At first no fluid escaped, but when a small bougie was passed, cerebrospinal fluid immediately dripped from the catheter at a rate of 40 to 50 drops a minute. No fluid was seen draining from the nose itself.

With the child in the lateral recumbent position, a unilateral suboccipital exploration was carried out through a straight lateral incision. The fracture line was immediately visualized, and while exposure was being obtained clear fluid could be seen welling into the field extradurally.

1. Ecker, A.: Cerebrospinal Rhinorrhea by Way of the Eustachian Tube: Report of Cases with Dural Defect in Middle or Posterior Fossa, J. Neurosurg. **4**:177-178, 1947.

The fracture line was widened into the mastoid and the exposed cells waxed. The dural tear was located adjacent to the internal auditory meatus. The surrounding dura was mobilized and the tear covered extradurally with a free graft of pericranium reenforced by absorbable gelatin sponge (gelfoam). Intradurally, the tear was covered with the same material. The seventh and eighth cranial nerves were visualized and found to be intact.

Postoperative Course.—The postoperative course was uneventful. The child remained afebrile, and rhinorrhea could no longer be demonstrated. During the 10 months which have elapsed since the operation there has been slow but satisfactory return of function of the damaged cranial nerves. She still shows slight hearing loss of mixed type on the right side. There has been no recurrence of rhinorrhea, and she has on two separate occasions contracted upper respiratory tract infections which cleared without complications.

SUMMARY

A case of cerebrospinal fluid rhinorrhea resulting from a traumatic craniomastoid fistula is reported. Preoperative identification of the site of leakage by catheterization of the eustachian tube enabled the correct surgical approach to be made and prevented a misdirected exploration of the anterior fossa.

153 Bishop Street (Dr. Ames).

Banner Building (Dr. Farmer and Dr. Mills).

TUMOR OF CAROTID BODY TYPE PRESUMABLY ARISING FROM THE GLOMUS JUGULARIS

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AND
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BOSTON

IN THE year 1743, von Haller first reported a gross description of a structure lying at the bifurcation of the carotid artery, which he termed the "ganglion minutum." The microscopic appearance of this organ was described by von Luschka in 1862. The first tumor of this body was surgically removed by Riegner in 1880 and described by Marchand in 1891. Since that time tumor of the carotid body has become a well known clinical entity, and over 250 cases have been reported in the literature to date. Guild,[1] in 1941, described a similar structure lying in the adventitia of the dome of the jugular bulb, immediately below the floor of the middle ear, which he called the "glomus jugularis." Guild [2] indicated that a glomus jugularis may be found at three sites, as shown in figure 1. Because of the size of the tumors in our two patients, it is impossible to be certain where they originated. In case 2 it is probable that the tumor originated in the jugular fossa. In 1945, Rosenwasser [3] published the description of a tumor of the carotid body type arising in the middle ear and mastoid process and suggested that it might have arisen from the glomus jugularis. Reports by LeCompte, Sommers and Lathrop [4] from this laboratory, and Kipkie,[5] in the recent literature, described similar tumors. Because the histories of two patients with tumors of the carotid body type, presumably arising in the glomus jugularis, are typical, and in the hope that additional knowledge concerning these tumors will aid in their diagnosis, these two cases are reported.

REPORT OF CASES

CASE 1.—A 26 year old housewife first noted tinnitus and gradually increasing deafness on the right side in Feb. 1943. Two months later she noticed diplopia, vertigo, nausea and vomiting and weakness and numbness of the entire left side of the body. In August 1943 she

From the Department of Neurosurgery, The Lahey Clinic (Dr. Poppen) and the Laboratory of Pathology, New England Deaconess Hospital (Dr. Riemenschneider).

1. Guild, S. R.: A Hitherto Unrecognized Structure, the Glomus Jugularis, in Man, abstracted, Anat. Rec. **79**:28, 1941.

2. Guild, S. R.: Personal communication to the authors.

3. Rosenwasser, H.: Carotid Body Tumor of Middle Ear and Mastoid, Arch. Otolaryng. **41**:64-67 (Jan.) 1945.

4. LeCompte, P. M.; Sommers, S. C., and Lathrop, F. D.: Tumor of Carotid Body Type Arising in Middle Ear, Arch. Path. **44**:78-81 (July) 1947.

5. Kipkie, G. F.: Simultaneous Chromaffin Tumors of Carotid Body and Glomus Jugularis, Arch. Path. **44**:113-118 (Aug.) 1947.

was seen elsewhere where two right temporal and postparietal burr holes were made and a brain abscess allegedly drained. There was no improvement following this procedure, and she was referred to the Lahey Clinic on Sept. 28, 1943. At this time there was marked nystagmus on lateral gaze, the right side of the palate was weak, there was minimal weakness of the right side of the face and hypesthesia and weakness on the left side. Roentgenograms at this time showed a difference in density of the petrous ridges on the two sides. Exploration of the right cerebellopontile angle was carried out, and a large vascular mass was found compressing the cerebellum and medulla on the right side. Some of the larger vessels were occluded by silver clips and by electrocoagulation, but because of the vascularity no further surgery was done and no biopsy specimen could be safely taken. It was thought that the mass was an angioma, and from Oct. 14 to Nov. 10, 1943, high voltage x-ray therapy was given, consisting of 1,800 r to each side of the skull. The patient improved slightly, and on Jan. 25, 1944, the vertebral artery was visualized by means of a thorotrast® injection and the right vertebral artery ligated. The angiograms showed some pooling of the dye in the region of the tumor. The patient noted

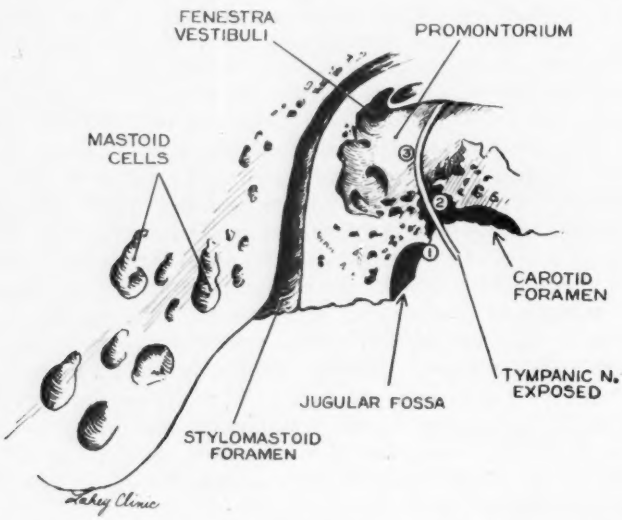

Fig. 1.—The areas in which a carotid body type glomus is likely to be found are indicated by the circles numbered *1*, *2* and *3*, according to the careful dissections made by Guild.

marked improvement and remained practically free of symptoms until 18 months before her last admission. At that time she began to have headaches and a bloody discharge from the right ear. Her physician cauterized a polypoid growth in the external ear. She was again admitted to this hospital on Sept. 26, 1947. There was a definite bruit over the right cerebellar defect. The hearing was diminished on the right side with bone conduction better than air conduction, and a grayish mass obstructed the external auditory meatus. Roentgenograms of the skull showed destruction of the right petrous ridge and apparent involvement of the mastoid process together with the large operative defect in the occipital region. The patient's condition failed and she died on Oct. 11, 1947, four years and eight months after her first symptom.

Postmortem Examination.—A complete autopsy was performed by one of us (P. A. R.), 1½ hours after death. The only findings pertinent to this report were found in the head and neck. Examination of the head revealed a bony defect in the right occipital region measuring 8 by 4 cm. In the right auditory canal there was a bluish red irregular mass reaching almost to the external

auditory meatus. Opening the calvarium revealed a large friable mass displacing the cerebellum and medulla to the left and adherent to the dura (fig. 2). This mass extended forward to the posterior clinoid processes but did not involve the sella turcica. It extended into the petrous ridge, destroying a large part of it, and filling the middle ear and mastoid cells. Inferiorly it extended down to the right side of the pharynx. A careful search of the neck failed to reveal any other masses along the jugular vein or carotid artery. The brain weighed 1,400 Gm. Coronal sections through the hemispheres revealed no changes with the exception of a uniform moderate dilatation of the ventricular system. The tumor mass involved the eighth, ninth, tenth, eleventh and twelfth cranial nerves on the right. The medulla was only a shell, because of the pressure exerted on it by the tumor. The intracranial portion of the tumor was encapsulated. Broad surfaces made by cutting the tumor showed a brown-gray spongy mass with a few calcareous deposits.

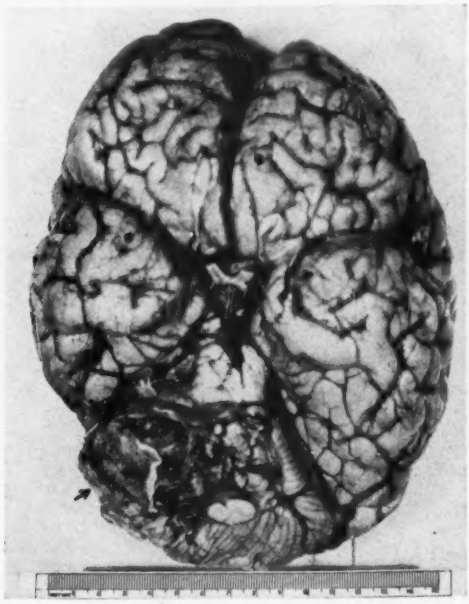

Fig. 2.—Intracranial extension of tumor mass into the right cerebellopontile angle.

Microscopic Examination.—Multiple sections revealed that the tumor was made up of sheets of epithelioid cells separated by a fine stroma (fig. 3). The cells were pseudosyncytial in appearance and showed twofold variation in shape and size. No mitotic figures were seen. The cytoplasm was clear and eosinophilic, with scattered brown granules. The nuclei were moderately pleomorphic, with granular chromatin, and occasional cells had one or two nucleoli. A few cells were multinucleated. The tumor was extremely vascular, containing many small blood vessels and other spaces lined by endothelial-like cells. The framework was reticular in character, stained with silver, and gave the tumor the typical compartmented appearance (fig. 4). In some sections collagen and in others bone was being invaded by tumor. There was intact dura lying between the tumor and the cerebellum.

Case 2.—A 63 year old woman entered the hospital March 11, 1948, complaining of dizziness, headaches, a pulsating feeling inside her skull synchronous with her pulse, periods of nausea and

a feeling of unsteadiness in the upright position as well as deafness in the right ear for the past year and a half. This patient had been operated on elsewhere three years previously, at which time the right common carotid artery was ligated because of a pulsating tumor mass immediately beneath and anterior to the right mastoid region. Two months later the patient began to have symptoms of a typical tic douloureux involving the right fifth cranial nerve, which was relieved by a supratentorial sensory root avulsion six months later. A loud bruit could be heard over the

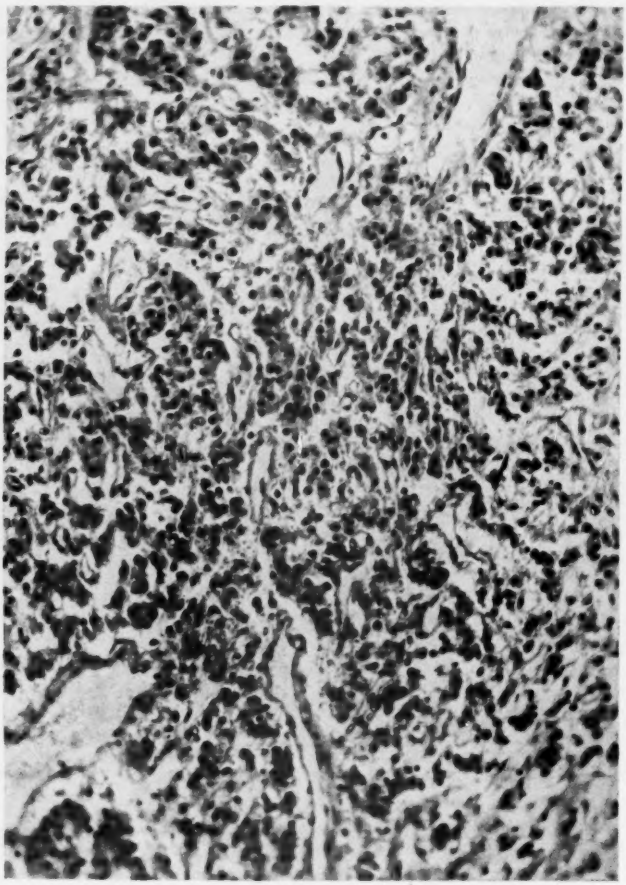

Fig. 3.—Tumor made up of sheets of epithelioid cells separated by a fine stroma; × 200.

right ear and mastoid region at that time. Roentgenograms of the patient's skull demonstrated erosion of the lateral portion of the temporal bone as well as the medial aspect of the mastoid tip (fig. 5). There was also erosion of the medial surface of the angle of the jaw. At this time, the mass could be seen extending into the posterior pharynx. In December 1947 the symptoms of dizziness and nausea increased, especially when the patient was in the upright position. Generalized headaches over the right frontotemporal region began at that time. The pulsations synchronous with her heart beat were also intensified; this was especially true in the right ear.

The partial deafness, previously noted, had progressed. In January 1948 the patient became unsteady on her feet and hesitated to walk alone for fear of falling.

At the time of her entrance to the hospital the patient was alert and cooperative. The blood pressure was 200 mm. systolic and 92 mm. diastolic. The patient complained of tenderness over the right temporal and mastoid regions. A large pulsating tumor mass could be seen immediately beneath the right ear and anterior to the mastoid. The tumor extended into the pharynx to such a

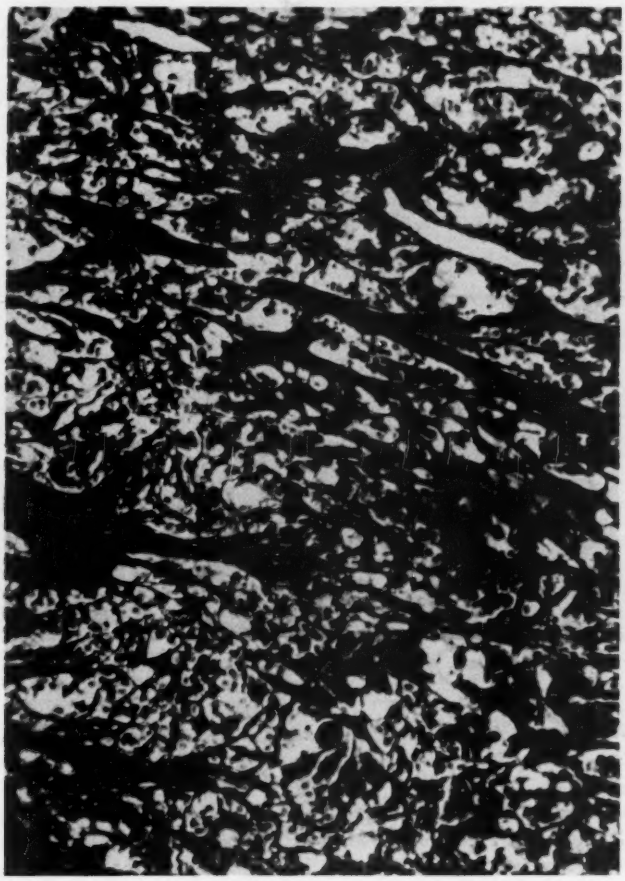

Fig. 4.—Typical compartmented appearance of tumor, stained with silver; × 122.

degree that it caused difficulty in swallowing and breathing. A loud bruit could be heard over the mass. Complete paralysis of the right fifth cranial nerve was present as a result of the previous sensory avulsion. There was a slight muscular weakness of the peripheral type in the right side of the face, and complete deafness of the right ear. The soft palate deviated to the left. The tongue was seen to protrude to the right, and there was paralysis of the right tenth nerve. The deep reflexes were uniformly exaggerated. There were no positive cerebellar signs.

It was thought that the patient had an aneurysm involving the right internal carotid artery and originating in the carotid canal. On March 25, 1948, the right internal and external carotid arteries as well as the jugular vessels were removed together with the tumor mass. The jugular vein was ligated at the jugular foramen and the internal carotid in the parasellar region. At the time of operation, the tumor was thought to be a carotid-body-like tumor, presumably originating in the glomus jugularis, since no extension was seen to the bifurcation and the carotid body itself seemed to be normal. Microscopic sections demonstrated the tumor to be of a carotid body type with metastases to five lymph nodes.

The patient did well after the operation. On the sixth postoperative day, however, a typical left-sided jacksonian convulsion occurred. Lumbar puncture disclosed a spinal pressure of 210 mm. The spinal fluid was clear and colorless and the total protein was 64 mg. per 100 cc. There were no further convulsive seizures, and the patient was discharged from the hospital on the eighteenth postoperative day with a diagnosis of tumor of the right jugular body with erosion of the petrous bone. The patient did well after her dismissal from the hospital; however, she died

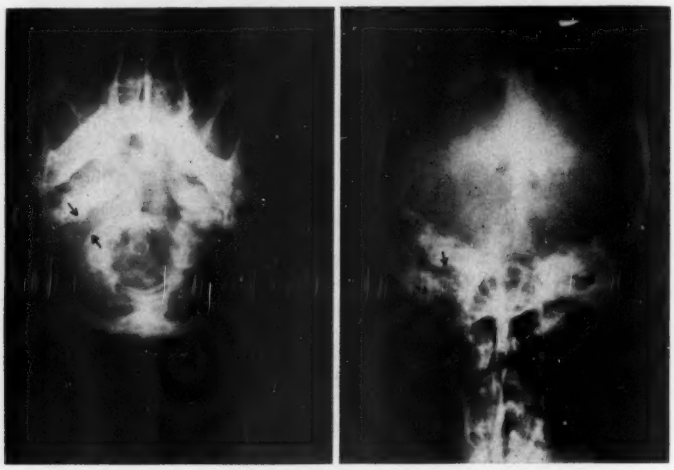

Fig. 5.—Erosion of base of skull on right in the region of the jugular foramen is clearly demonstrated.

on May 28, 1949, death being due to cerebrovascular accident resulting in a hemiplegia on the right side.

COMMENT

Descriptions of the histologic appearance of carotid body tumors are abundant in the literature.[6] This tumor, as well as those reported by Rosenwasser,[3] LeCompte, Sommers and Lathrop [4] and Kipkie,[5] presents the characteristic structure described by Rankin and Wellbrock [6c] as an alveolar perithelioma with whorls, or *Zellballen*. Because each presented a tumor in the middle ear and none showed any evidence of a primary tumor in the carotid body, the possibility of an origin from the glomus jugularis would appear to be sound.

6. (*a*) Gratiot, J. H.: Carotid Body Tumors: Collective Review, Internat. Abstr. Surg. **77**:177-186, 1943, in Surg., Gynec. & Obst. (Sept.) 1943. (*b*) LeCompte, P. M.: Personal communication to the authors. (*c*) Rankin, F. W., and Wellbrock, W. L. A.: Tumors of the Carotid Body: Report of Twelve Cases Including One of Bilateral Tumor, Ann. Surg. **93**:801-810 (April) 1931.

Certain points of similarity appear in all. Each had a history dating back over a relatively long time—in case 1 almost five years and in the others 10. It is interesting that the patient in case 1 was the youngest of the four at the onset of symptoms, and her youth may account for the rapidity of her course. All the patients late in the course of the disease presented a vascular growth in the external ear; increasing deafness and a discharge from the ear were frequent complaints. Each of the patients had had previous surgical procedures before the correct diagnosis was reached, and, because of the bleeding encountered and the gross appearance, the diagnosis of angioma or angiomatous polyp was entertained.

It is noteworthy that this patient (case 1) was given high voltage x-ray therapy for what was thought to be an angioma, with no relief; however, temporary relief did occur from a reduction of the blood supply by ligation of the vertebral artery. Bevan and McCarthy [7] reported a case of carotid body tumor in 1929 in which cure was obtained by x-ray therapy, but no other reports of relief in the treatment of carotid body tumors by x-rays or radium could be found. Therefore surgical excision, when possible, would seem to be the treatment of choice. It is possible with the high voltage x-ray equipment that roentgen treatment might be worth while.

Undoubtedly, other tumors of the middle ear which might fit this histologic picture have been described as endothelioma, hemangioma or perithelioma.

SUMMARY

Two patients with tumor of the carotid body type presumably arising in the glomus jugularis are presented, making a total of four available in the literature. This diagnosis should be considered for patients with a protracted history of increasing unilateral deafness, bloody discharge from the ear and vascular growth in the ear (a bruit was also noted in our patients), especially if roentgenograms show destruction of the petrous ridge and mastoid cells or if biopsy is accompanied with profuse bleeding. Adequate roentgen therapy may be of value in treating this condition. Early diagnosis with complete removal should be attempted.

NOTE.—Two additional cases of verified glomus jugularis tumor have been found since this report was completed.

7. Bevan, A. D., and McCarthy, E. R.: Tumors of Carotid Body, Surg., Gynec. & Obst. **49**:764-779 (Dec.) 1929.

OSSIFYING FIBROMA OF THE FRONTAL SINUS

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PHILADELPHIA

TUMORS involving the frontal sinus are frequently reported in the otolaryngological literature. These growths are usually benign in character, although malignant lesions are occasionally encountered. I believe that the otolaryngologist considers the commonest tumor involving the frontal sinus to be the mucocele; the rather uncommon encroachment of this sinus, the osteoma.

The interest in the case being reported was aroused by the difficulty in preoperative diagnosis. In view of the fact that biopsy reports established the diagnosis as that of ossifying fibroma, the literature was carefully investigated. Only one report was found of any similar lesion in otolaryngological journals. The standard textbooks devote no space to descriptions characterizing ossifying fibromas of the frontal sinuses.

On the basis of the pathology of frontal sinus osteomas, it is believed that the ossifying fibroma has its origin from either (*a*) an embryonic rest or (*b*) mucous membrane. The latter theory appears to be the more logical, because the surgical approach in this case revealed no evidence of any mucosal lining.

Ewing [1] states, "The exact point of origin of most fibromas is still undetermined. That many of them arise from misplaced islands of tissue, according to Cohnheim's theory, is very probable. . . . In the breast Williams found that 14 per cent of adenofibromas of this organ arise from embryonal rests, and the frequent association of chondroma and osteoma with fibroma, favors this view. For other fibromas one must apply the theory of local irritation and disturbance of nutrition as in keloid, which commonly follows scarring. . . Osteoma occurs as an element in many other tumors as a result of secondary ossification of the connective tissues."

The report of a case of ossifying fibroma of the ethmoid cells and frontal sinus by George H. Woodruff [2] in 1944 states that there did not appear to be a definite capsule. The tumor was somewhat fibrous in consistency though not very dense or very vascular. This case was first reported by the pathologist as, "Fibro-osteosarcoma, slow growing but malignant." Ten months after operation a small growth appeared in the upper anterior portion of the nasal cavity and on biopsy was found to be a fragment of sinus mucosa showing a polypoid hyperplasia on an inflammatory basis. At the same time in a review of the original sections another pathologist was of the opinion that it was not malignant. He stated, "I believe I would diagnose the original specimen as an ossifying fibroma type, but I doubt that it will metastasize."

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1. Ewing, J.: Neoplastic Diseases: A Treatise on Tumors, ed. 4, Philadelphia, W. B. Saunders Company, 1940.

2. Woodruff, G. H.: Ossifying Fibroma of the Ethmoid Cells and Frontal Sinus: Report of a Case, Ann. Otol., Rhin. & Laryng. **54**:582, 1945.

In a report of a fibroma of the ethmoid and frontal regions physicians Simpson, Graham and Sanders [3] made no mention of the ossification of this tumor. The lesion was a massive affair involving the left ethmoid and frontal sinuses and the nose and middle fossa dura and could readily have had its origin in the nasopharyngeal space.

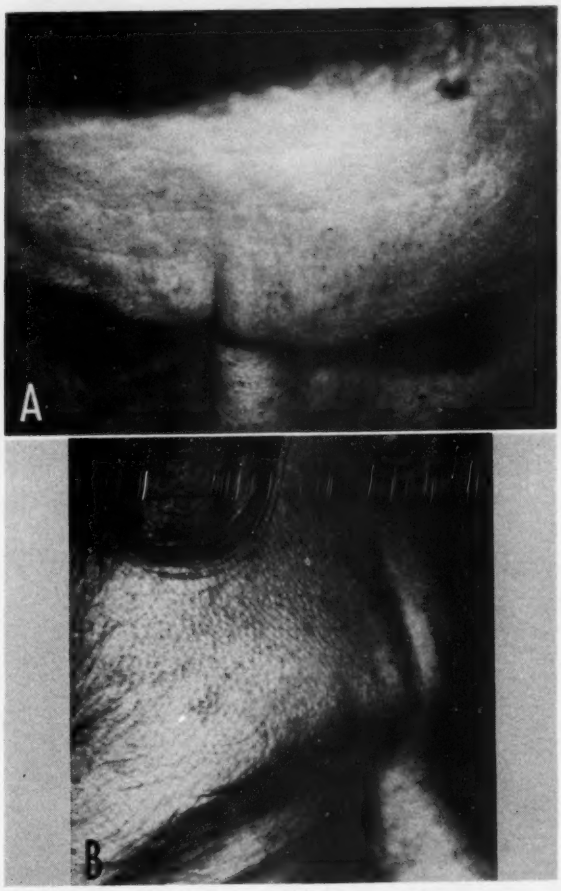

Fig. 1.—*A*, frontal view of patient revealing fulness of the left frontal area. *B*, lateral view of patient showing prominence of the left frontal region.

The diagnosis in the case to be reported presented both a clinical and a roentgenologic problem.

REPORT OF A CASE

M. C., a white woman aged 52, first visited me on Feb. 20, 1950 because of headaches for the past five years. These varied as to location, intensity and frequency but for the greater

3. Simpson, W. L.; Graham, D. G., and Sanders, S. H.: Fibroma of the Ethmoid and Frontal Region with Case Report, Ann. Otol., Rhin. & Laryng. **53**:344, 1944.

part were limited to the left frontal and vertex regions. The patient felt that her headaches had become greatly aggravated since the onset of menopause two years earlier. Three weeks prior to this visit she had noticed some fulness over the left frontal sinus. There were no associated ocular signs or symptoms.

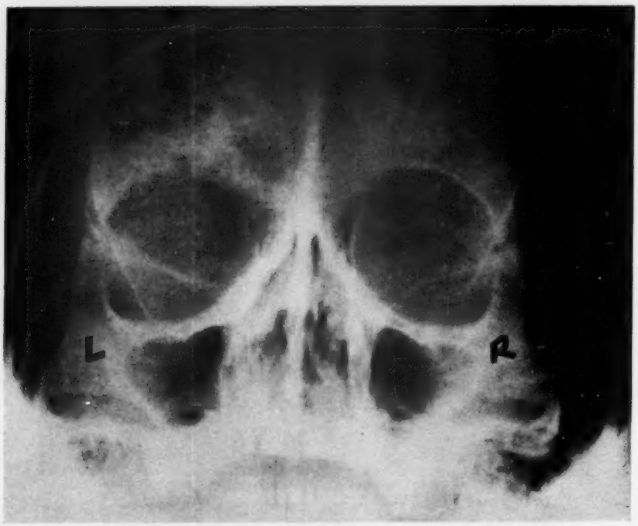

Fig. 2.—Mottled appearance of the frontal plate of the left frontal sinus.

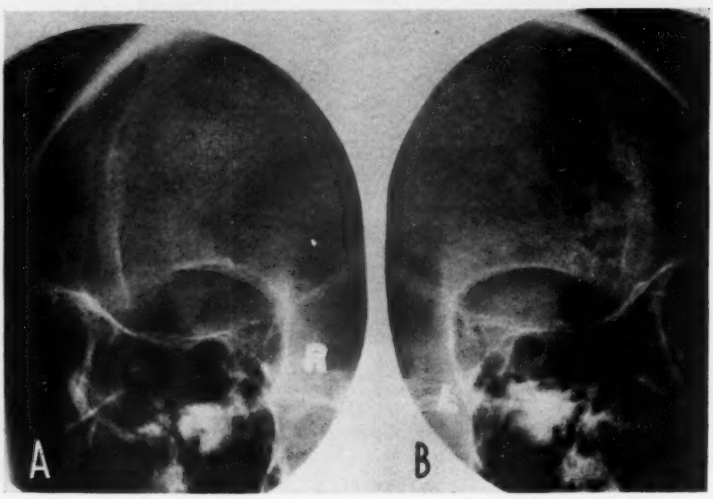

Fig. 3.—*A* reveals the normal right frontal sinus and *B*, the lesion in the left sinus.

Physical examination revealed a well developed and well nourished woman with slight fulness over the left frontal sinus. Her general physical condition was good. Examination of the nose presented no clinical evidence of sinus infection. The nasopharyngoscopic appearance was

normal in all respects. Both tympanic membranes were normal, but the external auditory canals revealed a bilateral external otitis media. Transillumination of the sinuses disclosed a very dense left frontal sinus, all the other sinuses being clear (fig. 1).

X-ray examination of the chest revealed no abnormalities. The ophthalmologic report showed the eyes to be normal, as were the blood cell count, urinalysis, basal metabolism and sedimentation rate.

The x-ray examination of the skull on March 2 demonstrated an ill defined, moderate-sized area of slight sclerosis interspersed with areas of bone destruction involving the anterior portion of the left frontal bone immediately adjacent to and above the left orbit. The roof of the left orbit was partially destroyed, and, in addition, the anterior portion of the left frontal bone was slightly thickened. Since I had no knowledge of the condition of the patient's chest or the results of serologic examinations the x-ray findings alone tended to favor an inflammatory rather than a malignant cause. This probably represented a fairly localized osteomyelitis which might have been syphilitic or nonsyphilitic in origin. It would have been surprising for this to rep-

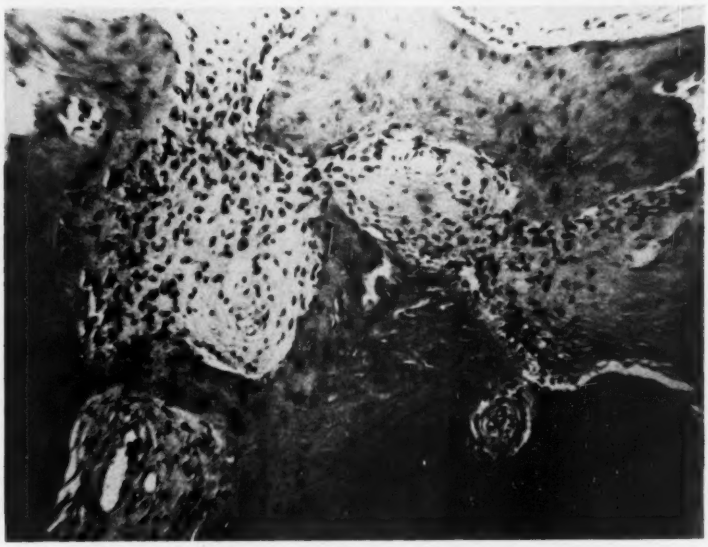

Fig. 4.—Area of predominant bone formation with relatively scarce fibrous tissue, approaching the pattern of a spongy osteoma.

resent a low grade pyogenic osteomyelitis, since there were no frontal sinuses present from which such an infection usually arises. Although a tuberculous osteomyelitis did not seem worthy of serious consideration, because of the rarity of this complication, the possibility of a metastatic malignant lesion had to be seriously considered, and an examination of the chest was recommended in an attempt to exclude either of the two latter possibilities. The smooth outline of the thickened outer table of the frontal bone tended to oppose a primary bone tumor cause, such as an osteogenic sarcoma. The sella turcica was normal in size and configuration, measuring 12 mm. in its anteroposterior diameter and 8 mm. in depth. The pineal gland was insufficiently calcified for identification. There was no evidence of abnormal intracranial calcification, and the results of the examination were otherwise essentially normal.

In summary, the examination of the skull demonstrated an area of minimal sclerosis interspersed with areas of bone destruction involving the anterior portion of the left frontal bone.

which probably represented a fairly localized osteomyelitis that might have been syphilitic or nonsyphilitic in origin. The possibility of a metastatic malignant lesion at this site had to be considered. However, an examination of the chest was recommended. The results of the examination of the skull were otherwise normal. The examination of the paranasal sinuses demonstrated minimal mucosal thickening involving the left frontal, right ethmoid and left maxillary sinuses. A moderate degree of mucosal thickening involved the left ethmoid sinus. The remaining sinuses were normal (figs. 2 and 3). The results of x-ray examination of the chest and long bones were reported as normal.

The patient was admitted to the Graduate Hospital of the University of Pennsylvania on March 6. Operation was performed on March 10. A modified Killian approach was used. Bleeding of the soft tissue, periosteum and bone was profuse. The anterior wall of the frontal sinus presented a bluish transparent appearance, giving the impression that there might be an angiomatous lesion behind it. This plate had been thinned as a result of pressure so that it

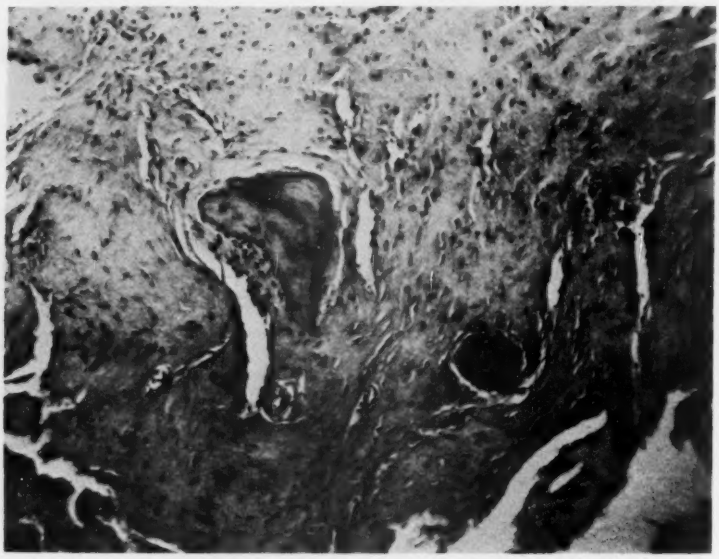

Fig. 5.—Predominance of relatively paucicellular, hyalinized fibrous tissue. This area approaches the appearance of fibrous dysplasia.

could readily be removed with tissue forceps. The space-taking mass which presented itself completely filled the sinus cavity. This was removed piecemeal by the use of a mastoid curet. Areas of both ivory-like and soft cancellous bone were encountered. The mass was broader along the floor of the sinus and extended from the intersinus septum to the lateral limits of the sinus cavity. The lesion was in intimate contact with the posterior wall, but there was no break in the bone and no dura exposed. It measured approximately 6 cm. by 3 cm. by 3 cm. As previously mentioned, there was no mucosal lining or encapsulating. The nasofrontal duct was not probed. Complete removal was accomplished so that a smooth-walled cavity remained. The incision was closed with a small drain.

The biopsy report was given on March 16 by Dr. A. Valdes-Dapena. The gross appearance was that of a great number of fragmented pieces of bone, varying from 0.5 to 1 cm. by 0.2 cm. by 0.4 cm. These pieces were pinkish white in color. The microscopic appearance was that of fibrous tissue poor in cells with various-sized islands of bone within it. This was benign

and was consistent with fibrous tissue replacing lesions of bone, the most likely diagnosis being ossifying fibroma (frontal bone) (figs. 4 and 5).

Recovery was uneventful, and the patient was discharged from the hospital on the seventh postoperative day. She has been under weekly observation for six months. The incision has healed with very little deformity. Headaches, which still persist, have lessened in severity and frequency. At no time has there been any intranasal evidence of sinus infection. The patient will be under observation for evidence of recurrence. The question of postoperative irradiation was considered, but the lesion is probably radio resistant and this therapy was not instituted.

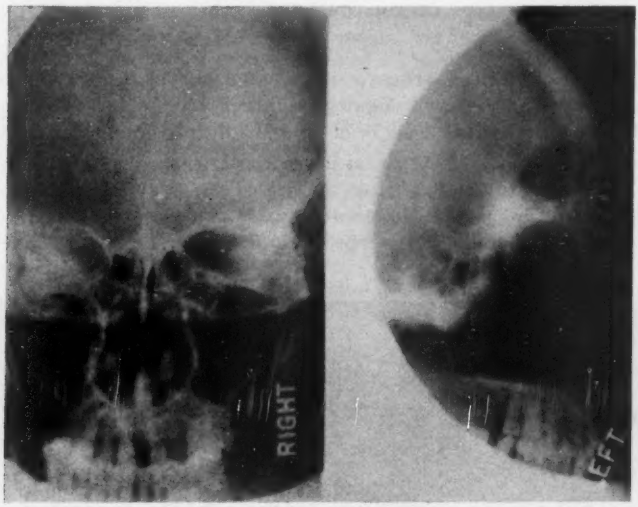

Fig. 6.—Anteroposterior and lateral x-ray views taken four months after operation showing the operative field in the left frontal sinus. No evidence of regrowth of the ossifying fibroma can be demonstrated.

A report of an x-ray examination made on July 11, approximately four months after surgical intervention, stated that there has been operative intervention in the region of the frontal sinus, left side. The density previously noted was removed, and the roentgenogram appearance was practically normal. The ethmoidal and maxillary sinuses showed no abnormality. Both orbits appeared normal. The mass which was previously noted could no longer be demonstrated (fig 6).

225 South Seventeenth Street.

Clinical Notes; New Instruments and Technics

PLASTER SKULL CAP WITH CURVED ARM BUMPER GUARD

Device for the Protection of Depressed Zygomatic (Malar) Bone Fractures Following Reduction

GERSON LOWENTHAL, M.D.
CINCINNATI

FREQUENTLY patients are seen who have recent depressed fractures of the zygomatic (malar) bone. Following reduction, which is relatively easy, it is feared that the fractures may again be displaced by the patient in his sleeping hours.

On several occasions I have found that a plaster skull cap with a curved arm bumper guard (figure) has protected the patient without the discomfort to him of sandbags about his head. The device is made either before or after reduction of the fractures. A stockinet is slipped over the calvaria and sheet wadding or

Plaster skull cap with curved arm bumper guard.

felt strips are then applied and a wire coat hanger is so bent that a curved arm projects over the zygomatic area involved without touching it. Padding is placed over this wire projection, and the entire ensemble is encased in plaster, using plaster bandages. During the day this headgear may be removed, and at night it is applied by the patient as a cap. The weight of the cap prevents the patient from tossing about too much in his sleep but still allows him to turn about freely to the affected side without dislocating the fractures.

This device is intended for the use only in the simple types of depressed fractures of the zygomatic area, in which external fixation is not necessary following reduction.

REPORT OF A CASE

J. T. C., a soldier, aged 35, was seen Aug. 7, 1945, five days after he had suffered a head injury when thrown against the dashboard in an automobile accident. There was no loss of consciousness. He complained of nasal obstruction on the left side and difficulty in chewing because of his inability to completely approximate his jaws.

From the Department of Otolaryngology, University of Cincinnati College of Medicine.

Presented as a new instrument at the Fifty-Fifth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 8, 1950.

Examination revealed a marked depression of the nasal bones, frontal processes of the maxilla, and of the zygoma on the left. The inferior orbital rim could not be palpated because of its depression and rotation into the antrum. A sharp, ragged fracture was palpable over the anterior antral wall. The left nasal fossa was almost occluded by depression of the lateral wall. Roentgenograms confirmed a diagnosis of depressed, comminuted fractures of the zygoma, frontal process of the maxilla and nasal bone on the left.

Under nitrous oxide, oxygen and ether anesthesia, the multiple (or combined) approach method was used. A heavy, straight elevator was inserted in the left nasal fossa, and the nasal bone and the frontal process of the maxilla were reduced by intranasal manipulation and elevation into position. An incision, about 1 cc. in length, was made in the buccogingival fold [1] over the left second molar area. A heavy, curved elevator was inserted close to the periosteum under the anterior root of the zygomatic arch and a pull exerted against the elevator. The zygoma was rotated laterally and the fracture of the anterior wall reduced. The inferior orbital rim was also somewhat elevated by this maneuver. One prong of a heavy towel clip was then engaged in the inferior orbital rim, which was elevated anteriorly and superiorly into position. One bleeding point was ligated, and the buccal wound was partially closed with one black silk suture. The left nasal fossa was packed lightly with petrolatum gauze.

A plaster skull cap in which a bumper guard was incorporated was fitted to the patient's head to prevent undue pressure on the left zygomatic area. Photographs in the figure show the plaster skull cap and the patient after reduction of the fractures.

280 Doctors' Building.

1. Kazanjian, V. H.: Injuries to the Face and Jaws Resulting from Automobile Accidents, Tr. Am. Acad. Ophth. **38**:275-308, 1933.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

OTITIS MEDIA AND COMPLICATIONS

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IN THE 1949 literature on otitis media and complications there are a large number of excellent articles, many of which should be studied in the original. The scarcity of good American articles noted last year is no longer evident.

ANATOMY

DeLisa [1] calls attention to the suture line separating the squamous part of the mastoid process and the petrosa. He pointed out that while the usual mastoid infection involves both portions, it is possible for either set of cells to be infected without the other. Infection of the squamous part alone would be marked by supra-auricular tenderness and probably by sagging of the superoposterior canal wall. Infection in the lower portion would produce tenderness in the lower part of the mastoid.

CHEMOTHERAPY AND BIOTHERAPY

Furstenberg [2] states that if improvement in the patient's condition with the use of penicillin is not definitely noted within 72 hours, it is likely that one is dealing with an organism resistant to the drug. Bacteriologic studies are too time consuming and costly to be routine.

"In spite of all available therapeutic measures today, the mortality of pneumococcic meningitis remains approximately 60%." Intrathecal administration is advised as well as intramuscular, since penetration of the meninges is poor with intramuscular administration. Aureomycin and chloramphenicol (chloromycetin®) administered by mouth give a concentration in the spinal fluid equal to 50 per cent of that in the blood. Furstenberg questions the value of any local use of the antibiotics in the nose or throat. In speaking of chronic otitis and sinusitis, he said, "I know of no authenticated instance of any of the antibiotics having cured a chronic suppurative otitis media or mastoiditis, or a chronic nasal accessory sinus disease, either when used systemically, locally, or by both methods." Nearly all acute infections of the ears, nose, sinuses and throat respond well to intramuscular administration of penicillin.

Moffett and Dalton [3] treated 17 children with acute mastoiditis with penicillin and sulfadiazine. Only one of these children required mastoidectomy, although all of these patients would have been operated on without delay before the advent of chemotherapy and biotherapy. All were admitted with swelling and tenderness over

1. DeLisa, D. A.: Squamositis, Mastoiditis and Squamomastoiditis, Arch. Otolaryng. **49**:558-563 (May) 1949.

2. Furstenberg, A. C.: Antibiotics in the Treatment of Diseases of the Ear, Nose and Throat, Ann. Otol., Rhin. & Laryng. **58**:5-17 (March) 1949.

3. Moffett, A. J., and Dalton, G. A.: Acute Mastoiditis in Children Treated with Penicillin and Sulphadiazine, Brit. M. J. **2**:1087-1089 (Nov. 12) 1949.

the mastoid, and in some cases there was actual fluctuation. Questionnaires were sent out 10 to 12 months after discharge from the hospital, and it was learned that only two patients had trouble later: One had an attack of earache four months later and another (the only one in which mastoidectomy was necessary) had an abscess under the scar four months later and recurring otitis media six months, eight months and again 11 months later.

Riskaer [4] (Copenhagen) treated 133 patients (with 195 affected ears) with penicillin and observed 127 patients (with 177 affected ears) as controls, using alcohol irrigations. Doses were 450,000 units daily for adults and smaller doses down to 150,000 units daily for infants under one year of age. Mastoidectomy was necessary in 11 per cent of the control series, and no operations were necessary in the penicillin-treated patients. The duration of suppuration averaged 7.1 days in the penicillin-treated patients and 13.2 days in the controls. Four cases of otitis media due to Hemophilus influenzae occurred in the group treated with penicillin; no improvement was seen in these patients. Penicillin seemed equally effective in the patients who came into the hospital late in the infection. However, patients who showed "manifest osteitis" on admission were not considered in this series. The administration of penicillin was continued three days after the discharge had stopped. In spite of this precaution there were 13 recurrences of infection in the penicillin-treated group, while there were eight recurrences in the untreated group. No symptoms of hypersensitivity were observed. (This is a well controlled study.)

Grebelius and Sjöberg [5] of Sweden report on 366 patients with acute otitis media seen in 1948 who were treated with penicillin. They chose as comparative material 329 cases from 1943, when penicillin was not available. They could not run parallel controls "because one cannot avoid giving penicillin to serious cases." The year 1943 was chosen as the comparative one because 1943 and 1948 were comparatively free from influenza epidemics. The condition in the patients treated with penicillin was serious enough for all to be admitted to the hospital. The results of cultures and sensitivity tests were reported. Only 2.1 per cent of the cultured organisms were insensitive to penicillin in vitro. Clinical results checked with the sensitivity tests. In several of the patients roentgenographic changes developed in the mastoids, thereby indicating necrosis, but with penicillin they cleared up without operation. One patient showed changes in the petrous apex without symptoms of apicitis. Roentgenographic observations were continued for five months before the calcium content started increasing. In 59 patients, 16.1 per cent, relapses occurred after apparent cure. Fifty of these were 3 years of age or less, and at this age level otitis has always been apt to reoccur. The authors therefore stress the importance of continuing use of penicillin for some time after the discharge has stopped, especially in small children. The average hospital stay for penicillin-treated patients was 8.0 days, and with sulfonamide treatment 12.7 days. With neither medication the stay averaged 15.9 days. The mastoidectomies required in 1948 were 2.5 per cent and 8.3 per cent in 1943.

They also reported that of 2,143 outpatients only 2.4 per cent required mastoidectomy, as compared to 8 to 20 per cent for the years 1938 to 1947. There was only

4. Riskaer, N.: Penicillin Treatment of Simple Acute Otitis Media, Acta oto-laryng. **37**:230-238 (June) 1949.

5. Grebelius, N., and Sjöberg, A.: Experiences of Penicillin Treatment in Acute Otitis Media, J. Laryng. & Otol. **63**:286-298 (May) 1949.

one death in the series, and it was that of an old woman who had meningitis and who was at the point of death on admission. No "masking-effect" was noted, and no allergic reactions were seen except for two cases of suspected "drug-fever." (This is another excellent paper, as are all of the Scandinavian articles.)

Thumin [6] reports a case of acute otitis media with no improvement after 10 days of medication with penicillin. Culture showed the organism to be a hemolytic Staphylococcus aureus (coagulase positive). Sensitivity tests showed the organism to be resistant to penicillin and sensitive to aureomycin. Treatment with aureomycin was begun, and the infection was controlled in another 10 days.

Local applications of streptomycin in concentrations of 5,000 to 25,000 units per cubic centimeter were used by Harris, Kilham and Finland,[7] of Boston City Hospital, in the treatment of chronic suppurative otitis media. Twenty-seven ears were treated and the results carefully tabulated. About 50 per cent of the patients had dry ears in a few days. Many of the organisms were resistant to streptomycin. In some cases the susceptible organisms quickly died, and then cultures showed heavy growths of the resistant bacteria.

(*Reviewer's Note.*—I have found that streptomycin powder diluted with boric acid powder [streptomycin one part to five parts boric acid] is very successful in the majority of cases of chronic otitis media, external otitis and draining radical mastoid cavities. The powder is excellent for office use; the solution used at home should help in certain cases.)

Parkes and Burtoff [8] report a case of a 32 year old Negro who had an acute otitis media and mastoiditis with a culture of Bacillus proteus. The symptoms were very acute, and blood cultures also revealed the same organism. The duration was only two days. Streptomycin was given intramuscularly, 0.5 Gm. every six hours. Within 48 hours the temperature was normal; in five days the discharge stopped, and the drug was discontinued on the seventh day.

Diehl and Morris [9] tried Sulfamylon (a sulfanilamide derivative) and streptomycin, 0.02 per cent, in the treatment of 29 cases of otitis media and external otitis. Good results were obtained in the cases of chronic otitis media, but no benefit was noted in those of external otitis or acute otitis media.

CHRONIC OTITIS MEDIA

Johnston [10] describes a study of chronic otitis in factory workers in England in which 1,902 workers were examined. Active middle ear infection was found in 5.3 per cent and external otitis in 2.5 per cent. The report is very complete, with many charts and graphs. Anyone interested in the English version of socialized medicine should read this article. The great difficulty in getting care for these infections is

6. Thumin, M.: Aureomycin in Penicillin-Resistant Acute Otitis Media, Laryngoscope **59**:1133-1134 (Oct.) 1949.

7. Harris, H. W.; Kilham, L., and Finland, M.: Local Streptomycin Therapy for Suppurative Otitis, Arch. Otolaryng. **49**:69-77 (Jan.) 1949.

8. Parkes, M., and Burtoff, S.: Streptomycin in Acute Hematogenous Mastoiditis Due to Bacillus Proteus, Ann. Otol., Rhin. & Laryng. **58**:260-262 (March) 1949.

9. Diehl, K. L., and Morris, A. G.: Sulfamylon and Streptomycin in Treatment of Infections of the Ear, Arch. Otolaryng. **50**:700-707 (Dec.) 1949.

10. Johnston, C. M.: The Incidence and Treatment of Infective Ear Disease in Factory Employees, Brit. M. J. **2**:1049-1059 (Dec. 18) 1948.

stressed. The time required for waiting in clinics is long. Not many of the patients would take the necessary time off from their work to care for what, to them, appeared to be a minor complaint. Many of the patients failed to complete treatment, even when the clinic was so set up as to make the time loss slight. Of the 213 patients who completed their treatments, 81 per cent were cured. Only 50 per cent of patients with radical mastoidectomy were found to have dry ears, and 77 per cent were severely deafened. Of 393 patients with severe deafness, the cause in 50 per cent was otitis media. The treatments consisted of irrigation with "spirit" (alcohol) and insufflation with antiseptic powders.

Rüedi,[11] of Switzerland, warns against the danger of the small marginal posterior perforations of the drum membrane. In his review of 351 cases of otitis media with cholesteatoma there were 11 with small posterior marginal perforations, in eight of which severe intracranial complications developed that were fatal in two. A mastoiditis in infancy with perforation at the margin is given as the cause. Epithelium grows into the fistula and causes a cholesteatoma. These small posterior perforations are much more rare and dangerous than the perforations of Shrapnell's membrane in the attic. In his series there were 121 cases with perforation of Shrapnell's membrane as compared to the 11 cases of the small posterior perforation.

Owen [12] treated 130 patients with chronic purulent otitis media with glycerite of hydrogen peroxide. In 106 patients of the 116 adequately treated, remissions of symptoms were obtained. The average time of treatment was three or four weeks and consisted of daily cleansing and application of the solution two to four times daily. There were no symptoms of irritation or toxicity.

"SILENT" MASTOIDITIS

Dolowitz and Wakefield [13] discuss a case of mastoiditis without otitis media. A 5½ year old boy was seen because of pain and swelling back of the left ear. The drum membranes appeared normal and hearing was normal. There had been no symptoms of otitis media for 2½ years. At that time he had been given sulfathiazole treatment for otitis media. Since that attack his growth and appetite had been poor. Operation showed a necrotic mastoid with subperiosteal abscess. The pus in the cavity was dry and resembled fecal material. Recovery was rapid, and his general health improved greatly after the mastoidectomy. The authors think the dormant infection lasted 2½ years and impaired the child's health during that time.

CHRONIC MASTOIDITIS

Baron [14] believes that the modified radical mastoidectomy should be done much oftener than the complete radical. In his last 10 cases, the complete radical operation was needed in only one, and in the other nine there were dry cavities, with the

11. Rüedi, L.: The Origin, the Clinical Importance and the Treatment of Small Posterior Marginal Defects of the Drum, Pract. oto-rhino-laryng. **6**:613-625 (Oct.) 1948.

12. Owen, W. E.: Treatment of Chronic Purulent Otitis Media with Glycerite of Hydrogen Peroxide, Laryngoscope **59**:1030-1035 (Sept.) 1949.

13. Dolowitz, D. A., and Wakefield, R. H.: A Case of Mastoiditis Without Apparent Otitis Media, Ann. Otol., Rhin. & Laryng. **58**:512-516 (June) 1949.

14. Baron, S. H.: Modified Radical Mastoidectomy, Arch. Otolaryng. **49**:280-302 (March) 1949.

development in most of them of better hearing after the operation than before. Baron believes in leaving the cholesteatomatous matrix in place in order to hasten the epithelization of the cavity. The way to cut two flaps from the skin of the canal is well shown in his illustrations. His review of the literature on the indications and technic of modified and radical mastoidectomy is very thorough.

Grimaldi, Mawson and Bateman [15] (England) present an interesting case of a woman aged 30 with long-continued otorrhea with recent vertigo and pain. A radical mastoid operation had been done nine years before, with a lateral sinus operation and jugular ligation a few days afterward. At operation a large cavity filled with cholesteatomatous material was found below the floor of the middle ear, extending backward deep to the facial canal. It was decided that the cavity was the space previously occupied by the jugular bulb. The authors remarked that the vertigo which was made much worse by preoperative swabbing of this area may have transmitted pressure to the aquaeductus cochleae and thus caused vertigo.

McGuckin [16] (England) has written an article based on "ideas emerging from an experience of nearly 200 cases coming to operation in the past 16 months." He is convinced that merely uncovering the pathological condition to allow drainage is not enough. The pathological site must be excised. During the past year he saw seven cases (the operation in four done by himself and in three by competent operators) in which there developed after operation involvement of the labyrinth or invasion of the oval window within a few months to a dozen or more years after the original operation. He remarks that Tumarkin's operation (transmeatal attico-antrostomy) was abandoned, after some success, because it did not provide adequate working space and did not leave space to treat the cavity afterward.

McGuckin observed several layers in biopsy specimens taken from the living tissue in the region of the inner wall of the aditus. There is first a layer of superficial cells of desquamating epithelium, next a layer of stratum granulosum with granulation tissue beneath. He has seen this granulation tissue continuous with deeper granulations beneath a layer of bone. This process, he thinks, is the type that goes on, unless radically removed, to erosions of the labyrinth even after apparently adequate radical operations. (His remarks on technic deserve study.)

Berg and Falkenberg [17] (Norway) did simple mastoidectomy operations in 15 cases of chronic otitis media with central perforations. The average duration of continuous preoperative discharge was seven years. Simple mastoidectomy was done in all cases. Cholesteatoma was not found in any case. Dry ears were obtained in all cases in an average of 17 days, but in only five of the cases were the ears dry when checked at eight to 12 months later. In six other cases there was improvement and in 3 there was not.

(*Reviewer's Note.*—It was not stated whether the operations were done by postaural or endaural technic or how closely the epitympanic areas were inspected for disease. It is possible that a modified radical technic could have been used with a higher percentage of dry ears and with preservation of hearing.)

15. Grimaldi, P. R. B.; Mawson, S. R., and Bateman, G. H.: A Case of Persistent Otorrhoea Due to Chronic Infection in a Large Infra-Tympanic Cavity, J. Laryng. & Otol. **63**:350-351 (June) 1949.

16. McGuckin, F.: Chronic Otitis Media, J. Laryng. & Otol. **63**:328-336 (June) 1949.

17. Berg, K., and Falkenberg, T.: Chronic Middle Ear Inflammation with Central Perforation: Surgical Treatment, Acta oto-laryng. **37**:415-419 (Oct.) 1949.

PATHOLOGY OF CHOLESTEATOMATOUS OTITIS MEDIA

Simonetta [18] (Italy) examined microscopic sections of many large cholesteatomatous masses (using the frozen section technic to avoid dissolving the crystals) and was unable to find cholesterin crystals. He then subjected two large specimens to chemical analysis. The trace of cholesterol was so minute as to be negligible. He found cholesterol crystals constantly in the type of condition he calls "cholesterinic otitis." In this type there is never the large mass of layered epithelial cells with the mother-of-pearl appearance. This second type is rarer and is characterized by a scanty, nonodorous discharge and the presence of a dense, yellow-brown exudate abounding in cholesterol crystals. The two types may occur together but are separate conditions. Cholesterol otitis is not to be confused with Christian's syndrome, which is characterized by granulomatous masses and is accompanied with changes in the cranial and facial bones.

TUBERCULOUS OTITIS MEDIA AND MASTOIDITIS

Siirala and Lahikainen [19] report five cases of tuberculous otitis media in which the use of streptomycin was successful in achieving a cure. Mastoidectomy was necessary in each case because of extensive bone destruction in the mastoid areas. Four of the patients were children and one an adult. The cases are reported in detail. One patient, a woman aged 21, had had drainage from the ear for six months and vertigo for two weeks. Radical mastoidectomy showed fistula of the horizontal semicircular canal. In spite of the fistula, Thiersch skin grafts were applied to the cavity, and the cavity healed as well as in nontuberculous radical cavities. The duration of streptomycin treatment varied from 10 to 19 days in the children and 48 days for the adult. The daily dose was 0.5 Gm. daily for the children for a total of 5 to 9.5 Gm. The adult received 1 to 2 Gm. the first few days, with a total of 43.5 Gm. in 48 days. No toxic effects of the drug were noted.

LATERAL SINUS THROMBOSIS

Hadfield and Hinde,[20] of England, report two cases of lateral sinus thrombosis, one with fatal termination in spite of the administration of large doses of penicillin and sulfadiazine. They were "published in order to illustrate the dangers of placing too much reliance on the antibiotics and chemotherapy." In the first case a simple mastoidectomy was done on a man aged 42 who had had an untreated draining ear for four months. A perisinus abscess and thrombosed emissary vein were found. The lateral sinus wall appeared thickened. The infection did not respond to antibiotics, and an operation on the lateral sinus was necessary 12 days later. The condition in the patient remained septic, and 10 days later a third operation revealed pus extending down along the lateral sinus to the jugular bulb. The patient finally recovered without ligation of the jugular vein.

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18. Simonetta, B.: Chronic Cholesteatomatous and Chronic Cholesterinic Otitis, Acta oto-laryng. **37**:509-515 (Dec.) 1949.

19. Siirala, U., and Lahikainen, E. A.: Streptomycin Treatment in Tuberculous Otitis Media, Acta oto-laryng. **37**:528-532 (Dec.) 1949.

20. Hadfield, E. H., and Hinde, R. T.: Two Cases of Lateral Sinus Thrombosis, J. Laryng. & Otol. **63**:605-609 (Oct.) 1949.

The second case concerned a boy of 15 years who had had a draining ear since infancy. There were symptoms of lateral sinus thrombosis on admission, but operation was delayed to see if antibiotics (which had not been used before) would control the infection. The boy died three days later. The organism was found to be B. proteus and was not sensitive to penicillin. (Chemotherapy and biotherapy are adjuncts and not alternatives to surgical procedure.)

Latella and Hopkins [21] describe a case of lateral sinus thrombosis affecting a 26 year old man in whom "sulfonamides and penicillin produced a masked picture of extensive mastoid breakdown and obliteration of the lateral sinus." The patient recovered after ligation of the jugular vein and radical mastoidectomy. In spite of accidental removal of the stapes, there were only four days of labyrinthine symptoms.

MENINGITIS

Foertsch and Fischer [22] report a case of pneumococcic meningitis affecting a man of 47. There had been no complaint referable to the ear except that several years before the patient had refused to go swimming because of some vague aural complaint. The patient was admitted in coma with pus in one auditory canal. Spinal tap and blood culture both showed type V pneumococci in large numbers. Modified radical mastoidectomy was done and penicillin given in a large intravenous dose with sodium sulfadiazine. One large dose of penicillin, 50,000 units in 3 cc. of water, was given intrathecally. The patient recovered with no bad effects on the central nervous system. (Smaller doses of penicillin intrathecally are usually advised because of possible tissue damage.)

Rudberg and Sjöberg [23] (Sweden) present a report on nine cases of meningitis, eight otogenic and one following acute frontal sinusitis. Five cases of meningitis developed after acute otitis media and three "in connection with a previously radically-operated ear." There was only one death, that of a 71 year old woman, moribund on admission. Penicillin given intramuscularly had no effect in their cases, but when given intraspinally improvement was definite. In one case not in this series, improvement was not noted until the penicillin was given by suboccipital puncture. It was assumed that partial obstruction by fibrin of the lumbar canal had occurred. In severe cases the intrathecal administration was given twice daily. In one case, that of an 18 year old girl, there was definite improvement of meningeal symptoms until the twice daily intrathecal injections of penicillin were cut to once daily. After a flare-up of symptoms, the dose was again given twice daily and the patient again improved and recovered.

Novotny,[24] of Vienna, states there is a type of otitic meningitis with a slow insidious onset very different from the usual abrupt stormy illness. It usually occurs in adults and rarely produces a high fever, the chief symptom being a dull headache with a feeling of pressure in the head. In five years 100 cases of otitic meningitis were observed at the Vienna Ear Clinic. Sixty-six were the usual type,

21. Latella, P. D., and Hopkins, J. H.: Lateral Sinus Thrombosis: Review of Literature and Report of Case, Ann. Otol., Rhin. & Laryng. **58**:147-158 (March) 1949.

22. Foertsch, J. H., and Fischer, A. J.: Pneumococcic Meningitis, Arch. Otolaryng. **49**:637-642 (June) 1949.

23. Rudberg, R., and Sjöberg, A.: On Prognosis and Therapy in Oto-Rhinogenous Meningitis, J. Laryng. & Otol. **63**:446-452 (Aug.) 1949.

24. Novotny, O.: Otogenous Meningitis, Monatsschr. f. Ohrenh. **82**:487-494, 1948.

and in 19 death occurred. In 34 cases the symptoms were so slight that lumbar puncture was necessary to make the diagnosis; 23 of the 34 were labyrinthine, 5 petrous, 3 postoperative and 3 tympanic in origin. Treatment comprised operation followed by chemotherapy and the use of antibiotics.

Skoog [25] found 20 cases of delayed meningitis occurring after skull fracture which involved the labyrinth. The delay in some cases was several years. In all cases there had been acute otitis media immediately after the injury. Operation was recommended to give better scar tissue closure between the middle ear and the labyrinth.

Savolainen [26] (Finland) found yeast fungi in the spinal fluid of five patients with otological and 11 with rhinological conditions with symptoms of meningitis. These patients were seen in the course of two years. In 12 of the 16 patients were found other organisms such as streptococci, pneumococci or staphylococci. After animal inoculations and other tests, the impression was gained that, although some yeast fungi may be toxic, most of these invaders were saprophytic rather than pathogenic. These yeast fungi were found in 20 per cent of all recognized cases of meningitis.

ENCEPHALITIS

Borries [27] (Denmark) again reminds us that encephalitis is not necessarily a stage of infection in its progress to brain abscess but is a separate entity. Since the symptoms of the two conditions are so similar, his observations on diagnosis are of value: "A very acute onset with high fever and marked disturbances of the sensorium, especially in the course of an acute otitis, is much more suggestive of encephalitis than of brain abscess." Of 73 patients only five died. "Even in apparently hopeless cases with aphasia, hemiplegia and coma, the patient may recover; . . . as a rule the patient is completely restored."

(*Reviewer's Note.*—Few otologists have seen enough of these cases to be competent in their diagnosis. Close cooperation with the neurologist is necessary.)

BRAIN ABSCESS

Shuster and Marks [28] report the case of a 12 year old boy who was brought to the hospital in a stupor. He had had an intermittent drainage of the right ear for six years. After a simple mastoidectomy, a cannula was passed into the temporosphenoidal lobe and 30 cc. of thin pus evacuated. The organism was Streptococcus hemolyticus. The patient recovered with no residual effects.

Wilmot [29] (England) presents a report on an airman who had had drainage from the right ear for nine months following swimming. He also had chronic pansinusitis on the right. While in the hospital under treatment for conditions

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25. Skoog, T.: Über die Spätmeningitis bei Labyrinthfraktur, Acta oto-laryng. **75**:137-143, 1949.

26. Savolainen, T.: On the Presence of Yeast Fungi in the Cerebrospinal Fluid in Oto- and Rhinogenic Meningitis, Acta oto-laryng. **37**:339-346 (Aug.) 1949.

27. Borries, G. V. T.: Otogenic Non-Purulent Encephalitis, Acta oto-laryng. **37**:483-497 (Dec.) 1949.

28. Shuster, B. H., and Marks, M. A.: Recurring Mastoiditis and Abscess of Right Temporosphenoidal Lobe, Arch. Otolaryng. **50**:462-465 (Oct.) 1949.

29. Wilmot, T. J.: Cholesteatoma Causing Cerebellar Abscess, J. Laryng. & Otol. **63**:682-684 (Nov.) 1949.

affecting his ear and sinuses, there developed headache, papilledema, nystagmus and incoordination of motion in the right arm and leg. At mastoid operation, a large cholesteatoma was found and the cerebellum approached through the mastoid cavity. An exploratory needle encountered 7 to 10 cc. of foul pus, from which coliform bacilli were cultured. A drainage tube was left in place and the cavity irrigated with penicillin solution for eight days. Penicillin was also given intramuscularly. The patient recovered with no after-effects.

Medwick, Uihlein and Hallberg [30] describe six cases of cerebellar abscess observed at the Mayo Clinic in the last 10 years. By close cooperation with the neurosurgeons, there was recovery in four of the six cases. In one of the cases cerebellar symptoms developed after an acute otitis during influenza, but in the other five these symptoms developed after chronic otitis media of months' or years' duration. The case histories are given in detail and should be studied. (Although such complications develop infrequently, a familiarity with the various symptoms should be gained by study of such reports.)

PETROSITIS

Hörbst [31] discusses a very interesting case of petrositis occurring in a woman of 30 years who had had chronic otitis media for several years with occasional headache and earache. A radical mastoidectomy was done. Later, there developed paralysis of the sixth nerve and deep pain suggestive of petrositis. A second operation was done, and a cholesteatomatous mass was found in the attic together with a few diseased cells around the labyrinth. The symptoms, including paralysis of the sixth nerve, began again after influenza, and a third operation was done with Ramadier's technic. In the tip of the petrous process was found a small cavity filled with granulation tissue but no free pus. The patient recovered.

Berggren [32] describes a case of metastatic brain abscess and petrositis developing after an abscess of the jaw in a man aged 45. There had been no otitis media. The patient died of heart disease, and autopsy disclosed the brain abscess and an area of infection in the petrous tip. The symptoms had included paralysis of the sixth nerve and some loss of hearing.

Nakó [33] (Budapest) reports a case of petrositis occurring in a man aged 24. He had had numerous ear infections in the past that produced much bony sclerosis. A search for a tract of cells leading into the petrous area was not successful, and penicillin was given after definite signs of meningitis began. The patient recovered in spite of the fact that the focus in the petrous pyramid was not drained.

Lidén [34] (Sweden) presents a report concerning a 37 year old man with a pharyngeal abscess developing secondary to a mastoiditis and petrositis. Simple

30. Medwick, J. X.; Uihlein, A., and Hallberg, O. E.: Abscess of the Cerebellar Lobe of Otogenic Origin, Arch. Otolaryng. **50**:429-439 (Oct.) 1949.

31. Hörbst, L.: Die Pyramidenspitzenerkrankung bei chronisher Otitis media, Monatsschr. f. Ohrenh. **83**:77-83, 1949.

32. Berggren, S.: Fall av metastatisk och hjärnabscess, Nord. med. (Stockholm) **38**:1038-39, 1948.

33. Nakó, A.: Recovery from Petrositis Associated with Meningitis After Penicillin Treatment, Acta otō-laryng. **37**:153-160 (April) 1949.

34. Lidén, von G.: Otogene Pharynxabscesse, Acta oto-laryng. **37**:108-115 (April) 1949.

mastoidectomy was done a month after spontaneous rupture of the drum membrane. Symptoms of petrositis developed, and a month later the petrous pyramid was opened and drained with the technic of Streit. Shortly afterward a parapharyngeal abscess developed. After drainage, the patient recovered. A roentgenogram showing involvement of the petrous pyramid illustrates the article.

LABYRINTHITIS

Rosenwasser [35] discusses acute diffuse labyrinthitis and reports two cases with recovery in both. The first case concerned a man aged 26. Two days after the onset of an acute otitis media there developed nausea, vomiting, severe headache and a temperature of 103 F. Examination showed constant horizontal nystagmus toward the opposite ear. With the Bárány noise apparatus in the good ear no hearing could be demonstrated in the infected ear. Medication consisted of crystalline penicillin, 500,000 units administered intramuscularly every three hours, and intravenous injection of sulfadiazine, 1 grain per pound of body weight, daily. Medication was continued three weeks, at which time all symptoms had disappeared and the hearing had returned to normal. The labyrinth also reacted normally on testing. The second case concerning a child aged 10 years, was not observed so early, and in spite of the same intensive medication the infected labyrinth remained inactive and hearing was lost completely. In the first case the infecting organism was Staphylococcus albus B and in the second, Pneumococcus type XII. Rosenwasser thinks it is possible that the permanent damage in the second case may be due partly to the fact that the organism was type XII and partly because the patient was not admitted to the hospital until the third day of his illness. He suggests that larger doses of penicillin be used in serious cases.

OTITIC HYDROCEPHALUS

Horowitz,[36] of Glasgow, reports in great detail three cases with severe intracranial hypertension of the type frequently referred to as otitic hydrocephalus. He proposes the term otogenic intracranial hypertension. The name otitic hydrocephalus was suggested by Symonds in 1930 because ventricular taps produced a copious flow of cerebrospinal fluid. Encephalograms, however, show no distention of the ventricles. In the cases reported were signs of greatly increased intracranial tension coming on slowly, over a period of weeks. Headaches, nausea, vomiting and swelling of the optic disks were characteristic. Lumbar puncture showed pressures from 380 to 520 mm. of water. The condition in the first case developed after bilateral ligation of both jugular veins; ligation was necessary because of multiple emboli that produced skin lesions.

In the second case symptoms developed two weeks after packing of the lateral sinus, which had been injured during mastoid operation. The third case concerned a girl of 14 in whom a lateral sinus thrombosis developed that required packing of the sinus and ligation of the jugular vein. Two weeks later symptoms of intracranial pressure developed followed by symptoms of petrositis. Drainage of the

35. Rosenwasser, H.: Two Cases of Acute Diffuse Labyrinthitis Following Acute Otitis Media, Laryngoscope **59**:345-353 (April) 1949.

36. Horowitz, S.: Otogenic Intracranial Hypertension, J. Laryng. & Otol. **63**:363-381 (July) 1949.

apex was not necessary. In two cases symptoms of pressure were controlled by frequent lumbar puncture; in the other case a subtemporal decompression was done to relieve pressure that was causing impairment of the vision. Vision came back to normal in all three cases. Horowitz concludes that although the cause of the hypertension is still problematic these cases support the theory that the condition is most likely brought about by obstruction to the outflow of venous blood from the cranial cavity.

Smith and Mirmelstein [37] discuss a case of intracranial pressure following a simple mastoidectomy in a man aged 27. Operation was done because of a flare-up of pain and discharge that had been intermittent for seven years. There was partial thrombosis of the lateral sinus, and the internal jugular vein was ligated. The spinal fluid pressure remained high, and five months after the mastoidectomy a brain surgeon did a subtemporal decompression and punctured the brain in five directions in the search for a brain abscess. Air was then injected into the ventricle to rule out suspected brain tumor. A decompression was done, and a diagnosis of otitic hydrocephalus was then made. The authors agree with Williams that the term otitic hydrocephalus is not completely accurate but may help to keep otologists on the alert for its presence with otitis. The patient was discharged free from symptoms after eight months. A year later he was still symptom free.

McGovern [38] reports a case of "otitic hydrocephalus" that developed after a mild otitis media in a boy aged 6 years. Total duration of the illness was only three weeks. Mastoidectomy was performed because of intense headaches and papilledema of 3 diopters. The lumbar puncture showed the fluid to be normal but under intense pressure. The operation disclosed a perisinus abscess with granulations covering the lateral sinus. The lateral sinus was not opened, and the patient recovered rapidly, leaving the hospital free from symptoms referable to the eye seven days after operation.

Brunner [39] discusses the occurrence of otogenous edema of the brain in two children, one aged 10 and the other 1½ years. In both of these children acute symptoms of intracranial irritation developed after aural infections of short duration. In the first child aphasia developed and a one-sided epileptiform attack. Papilledema was absent, and there was no increase in pressure of the spinal fluid. After simple mastoidectomy three punctures of the brain were done in search for an abscess. No pus was found. The condition cleared up in about three weeks. In the second case the symptoms pointed to involvement of the cerebellum. The cerebellum was exposed and punctured. No brain abscess was found. Recovery was rapid and complete in a few weeks.

(*Reviewer's Note.*—The cases reported by Brunner and by McGovern are not the same type described by Symonds in which the symptoms of pressure persist for months. They may be the type described by Borries as acute encephalitis.)

37. Smith, A. T., and Mirmelstein, A.: Otitic Hydrocephalus, Ann. Otol., Rhin. & Laryng. **58**:275-279 (March) 1949.

38. McGovern, F. H.: Otitic Hydrocephalus, Eye, Ear, Nose & Throat Monthly **28**:122-125 (March) 1949.

39. Brunner, H.: Otogenous Edema of the Brain, Ann. Otol., Rhin. & Laryng. **58**:997-1012 (Dec.) 1949.

CHRONIC SECRETORY OTITIS

Hotchkiss [40] reports observing about 30 cases of the serous otitis which have lasted from six months to 3½ years. In one persistent case he did a mastoidectomy and found the cells normal in appearance except that they were filled with yellowish serum. The symptoms did not improve after operation. In a few of his older patients he has intentionally made permanent openings in the tympanic membrane with improvement in their hearing. His usual treatment is incision of the drum membrane and removal of the fluid by suction. He uses inflation with hesitation and for diagnosis only, fearing to start secondary infection and produce damage to the eustachian tube. (This interesting article was missed in last year's review; it agrees in the main with Tobey and others who have written on the subject.)

TECHNIC

Lempert's [41] article on the endaural operation for chronic otitis media is very well illustrated with 13 figures. His incision removes a large flap of skin from the posterior canal wall, which is saved to use as a skin graft for the postoperative area. He no longer uses the perforating drill to go blindly down to the mastoid antrum but removes the posterior wall with a medium-sized burr, exposing the incus at once and then working backward to remove any antral or mastoid site of the pathologic process. With careful complete removal of all such sites a dry middle ear should be obtained.

House [42] describes his technic of operating for chronic suppurative otitis media. There are 12 excellent illustrations in the article. He advises identification of the horizontal canal and facial ridge before cleaning out the middle ear, since what the operator sees he will not injure. (This article and the preceding one should be studied for the many valuable details.)

Daggett [43] describes a modified radical mastoid operation in which no flap is cut. He advises that if the incus is diseased, and in his experience it is diseased in almost all cases, it should be removed along with the head of the malleus. This removal of the head of the malleus makes it easier to clean out the attic and gives a better chance for the upper part of the drum to seal itself off from further drainage from above. He advises that if the canal is narrow or if there is a tendency toward external otitis the usual flap should be made. His results in 40 cases are satisfactory. The advantages are absence of painful dressings and simplicity of after-treatment. He uses the postauricular incision and says the majority of English surgeons use this approach.

(*Reviewer's Note.*—As the author says, the omission of the metal flap is not new. It has some advantages in cases in which it is certain that all of the pathological site has been reached and removed. It is also advised by House in selected cases.)

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40. Hotchkiss, W. T.: Chronic Secretory Otitis, South. M. J. **41**:727-732 (Aug.) 1948.

41. Lempert, J.: Lempert Endaural Subcortical Mastoidotympanectomy for the Cure of Chronic Persistent Suppurative Otitis Media, Arch. Otolaryng. **49**:20-35 (Jan.) 1949.

42. House, H. P.: Surgery of the Chronically Discharging Ear, Arch. Otolaryng. **49**:135-150 (Feb.) 1949.

43. Daggett, W. I.: Operative Treatment of Chronic Suppurative Otitis Media, J. Laryng. & Otol. **63**:635-646 (Nov.) 1949.

Meurman and Ojala [44] (Finland) observed that in about 10 per cent of his 700 cases in which radical mastoidectomy was required the postoperative cavity was too large to keep clean easily. In those cases in which the pneumatization extended down toward the tip, a muscle periosteal flap was cut from the soft tissues. This procedure was done in 33 cases with the postauricular incision and through the endaural approach in three cases. A simultaneous skin graft was done in all of the endaural cases and in five of the postauricular cases. Contraindications are threatened intracranial complications and suppurative inflammation of the soft tissues.

MISCELLANEOUS

Handstedt [45] reports an interesting case of air embolus occurring during a mastoid dressing. A simple mastoidectomy was done on a 38 year old woman. During operation the lateral sinus was injured and severe bleeding required a tampon. The tampon was removed on the eleventh day while the patient was in a sitting position. A gurgling sound was heard, bleeding began and the patient fainted. The tampon was replaced. Roentgenograms showed increase in size of the right side of the heart and partial atelectasis of the right upper lobe of the lung. The patient complained of pain in the chest. Pain and the conditions observed roentgenologically disappeared in a few days, and the patient survived. "As possibility of a sinus thrombosis could not be excluded there was performed immediately . . . ligation of internal jugular vein" and inspection of the lateral sinus. No thrombus was found. He advises that "all manipulations with the sinus should be performed while the patient is lying down. He found five reports of air embolus during ear operations with three fatalities. Twelve patients with air embolus occurring during change of dressings recovered.

Johnsen [46] reports that in the Rigshospital, Copenhagen, electrocoagulation has been used in many cases in which the severer surgical operation of section of the fifth nerve (for severe neuralgia) has been contraindicated. A trocar was passed through the zygomatic region through the foramen wall. An electrode was passed through the trocar and electrocoagulation was performed. Among 74 patients that were observed in follow-up examination, seven had otitis media in one to three weeks; 20 per cent complained of symptoms indicating obstruction of the eustachian tube on the same side. Surgical severance of the nerve (retroganglionic) produced symptoms of impaired hearing in half the cases by Dederdig, who was cited by Johnsen. The surgical operation may partially or entirely cut the motor root of the third branch and produce paralysis of mastication of the jaw on same side. The electrocoagulation procedure always does this. This procedure probably causes paralysis of the tensor veli palatini, which is the only muscle by means of which the eustachian tube is opened, thereby affording a suppurative otitis media the chance to ascend.

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44. Meurman, Y., and Ojala, L.: Primary Reduction of a Large Operation Cavity in Radical Mastoidectomy with a Muscle-Periosteal Flap, Acta oto-laryng. **37**:245-252 (June) 1949.

45. Handstedt, S.: Air Embolism after Operation, Acta oto-laryng. **37**:57-64 (Feb.) 1949.

46. Johnsen, S.: Otitis Media Following Electrocoagulation of the Gasserian Ganglion After the Method of Kirschner, Acta oto-laryng. **37**:45-49 (Feb.) 1949.

News and Comment

CHANGE OF MEETING PLACE

The meeting place of the American Laryngological, Rhinological and Otological Society, the American Broncho-Esophagological Association, the American Laryngological Association and the American Otological Society, Inc., has been changed from White Sulphur Springs to the Claridge Hotel, Atlantic City, N. J., the week of May 6, the dates being the same as those given in the Directory of Otolaryngological Societies in this issue.

Directory of Otolaryngological Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. J. Milton Robb, 1553 Woodward Ave., Detroit 26.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.
Place: Atlantic City, N. J. Time: June 13-15, 1951.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Derrick Vail, 700 N. Michigan Avenue, Chicago 11, Ill.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

American Board of Otolaryngology

Secretary: Dr. Dean M. Lierle, University Hospitals, Iowa City.
Place: Hotel Biltmore, New York. Time: Jan. 8-11, 1951.
The John Marshall Hotel, Richmond, Va. Time: May 1-4, 1951.

American Broncho-Esophagological Association

President: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Place: The Claridge Hotel, Atlantic City, N. J. Time: May 7 and 8, 1951 (p. m.).

American Laryngological Association

President: Dr. Gordon B. New, 102 2d Ave., S.W., Rochester, Minn.
Secretary: Dr. Louis H. Clerf, 128 So. 10th St., Philadelphia 7.
Place: The Claridge Hotel, Atlantic City, N. J. Time: May 9-10, 1951.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Louis H. Clerf, 128 So. 10th St., Philadelphia 7.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: The Claridge Hotel, Atlantic City, N. J. Time: May 6-8, 1951 (a. m.).

SECTIONS:

Eastern.—Chairman: Dr. Harry P. Schenck, 326 So. 19th St., Philadelphia 3.
Place: Philadelphia. Time: Jan. 12, 1951.
Southern.—Chairman: Dr. Charles C. Grace, 145 King St., St. Augustine, Fla.
Place: Miami, Fla. Time: Jan. 17, 1951.
Middle.—Chairman: Dr. Clarence W. Engler, 2323 Prospect Ave., Cleveland.
Place: Cleveland. Time: Jan. 15, 1951.
Western.—Chairman: Dr. Lewis F. Morrison, 490 Post St., San Francisco.
Place: San Francisco. Time: Jan. 27-28, 1951.

American Otological Society

President: Dr. Kenneth M. Day, 121 University Place, Pittsburgh.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago.
Place: The Claridge Hotel, Atlantic City, N. J. Time: May 11-12, 1951.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Norman N. Smith, 291 Whitney Ave., New Haven 11, Conn.
Secretary: Dr. Joseph G. Gilbert, 111 E. 61st St., New York 21.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. George E. Shambaugh Jr., 55 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Joseph Hampsey, 806 May Bldg., Pittsburgh 22.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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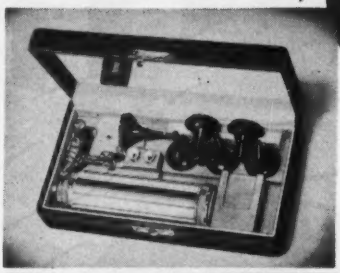

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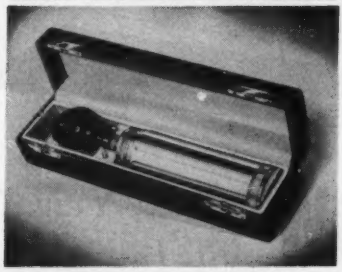

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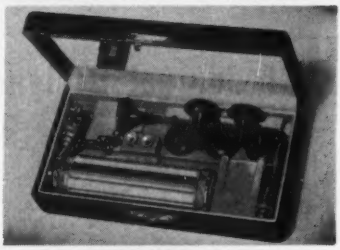

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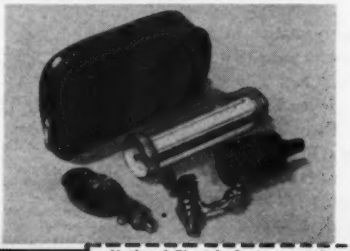

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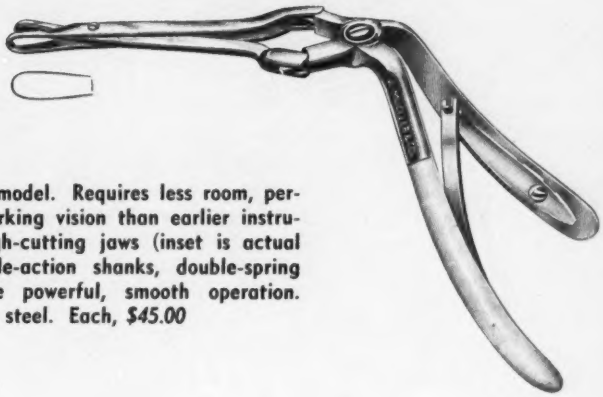

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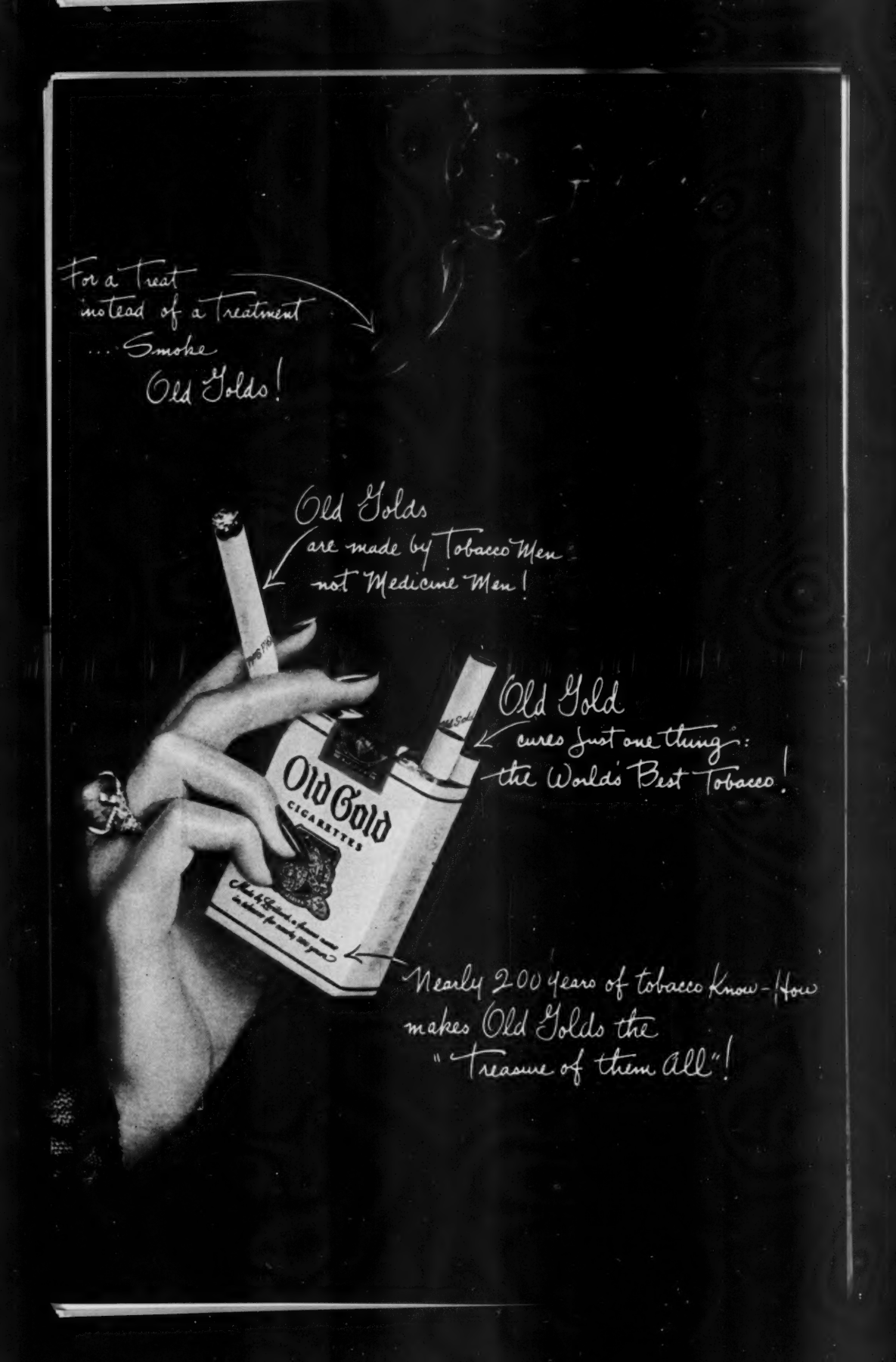
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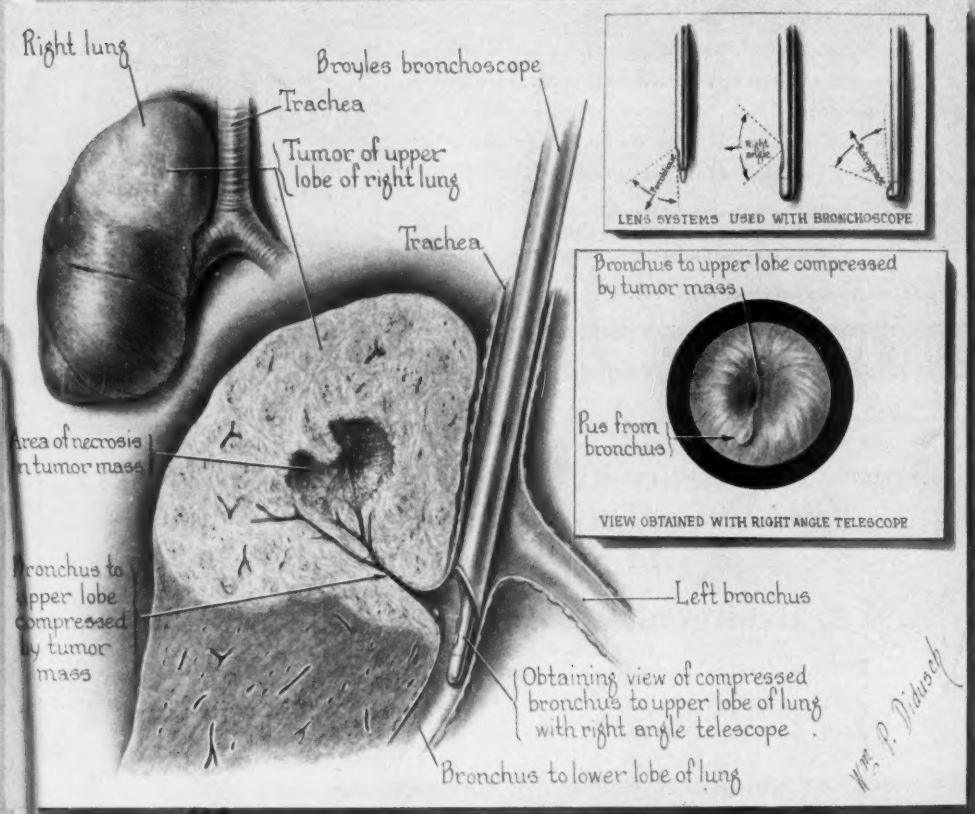
Right lung
Broyles bronchoscope
Trachea
Tumor of upper lobe of right lung
Trachea
Area of necrosis in tumor mass
Bronchus to upper lobe compressed by tumor mass
Left bronchus
Obtaining view of compressed bronchus to upper lobe of lung with right angle telescope
Bronchus to lower lobe of lung
LENS SYSTEMS USED WITH BRONCHOSCOPE
Bronchus to upper lobe compressed by tumor mass
Pus from bronchus
VIEW OBTAINED WITH RIGHT ANGLE TELESCOPE

The illustration shows a case of suspected tumor of upper lobe of right lung in which x-ray findings were inconclusive. The Broyles Bronchoscope with right angle lens system disclosed a compressed bronchus, and a diagnosis of tumor was made.

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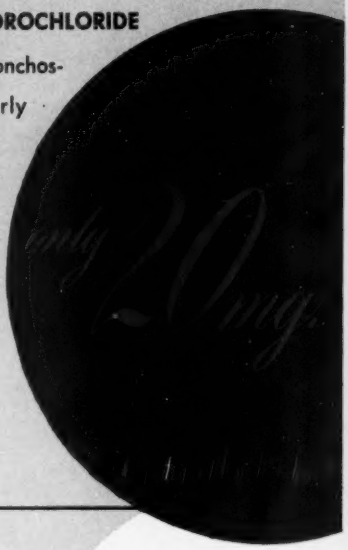

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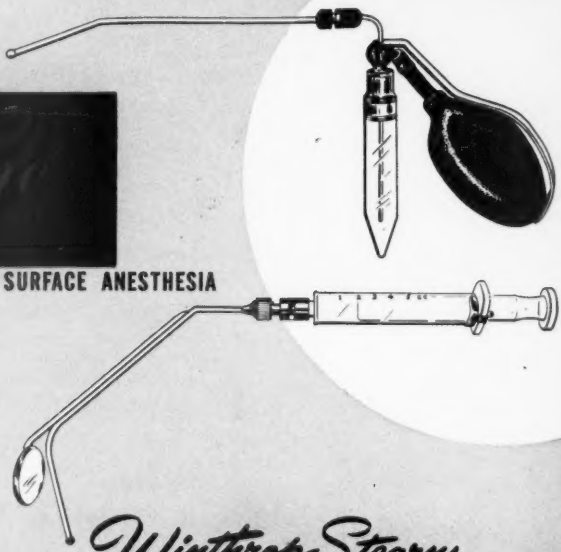

1. Carabelli, A.: *Diseases of the Chest*, 15:532, May, 1949.
2. George P. Pilling & Sons, Company, 3451 Walnut Street, Philadelphia 4, Pa.

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